1966

Prediction of genetic improvement in a finite population under selection

Kenneth Eugene Rowe
Iowa State University

PREDICTION OF GENETIC IMPROVEMENT IN A FINITE POPULATION UNDER SELECTION

by

Kenneth Eugene Rowe

A Dissertation Submitted to the

Graduate Faculty in Partial Fulfillment of

The Requirements for the Degree of

DOCTOR OF PHILOSOPHY

Major Subject: Animal Breeding

Approved:

Signature was redacted for privacy.

In Charge of Major Work

Signature was redacted for privacy.

Head of Major/Department

Signature was redacted for privacy.

Dean of Graduate College

Iowa State University
Of Science and Technology
Ames, Iowa

1966

TABLE OF CONTENTS

INTRODUCTION

The increased understanding of principles of genetic improvement has been a gradual process of integrating observed results with the development of theory. The mathematical formulation of theory is necessarily limited to the consequences of the assumptions made. The complexity of any biological system precludes anything like complete generalization. In recent years, numerical methods have been used in quantitative genetics research to bridge the gap between theory and the complexity of the actual biological system. For example, high-speed computers have been used to simulate populations whose members are carefully specified in terms of certain aspects of the manner in which a biological organism might function, even though such a specification never attains the full complexity of an actual biological population. Such populations are useful in at least two ways:

(1) To enable the researcher to investigate the consequences of genetic systems too complex to yield to mathematical treatment, and

(2) To confirm theory already developed.

Many aspects of genetic systems are expected to behave in a manner which is expressible in terms of the laws of probability and may therefore be simulated through the use of random numbers. Thus, this simulation technique has been called Monte Carlo investigation.

In general the methods used to predict genetic improvement in

populations, which are necessarily finite, are based upon theory developed from mathematical formulations requiring that certain simplifying assumptions be made to facilitate their solution. Simulation should be useful in examining the consequences of relaxing some of these limiting assumptions. Resemblance between relatives has been and is being used in methods of predicting genetic improvement. This is possible because the expectations of the covariances, in terms of components of hereditary variance, are known if certain assumptions can be made. The mathematical formulation of the genetic expectations of the covariances between relatives, and thus the correlations between relatives, has been impossible without making some assumptions which may restrict their utility for predictive purposes. Some of the assumptions which are unrealistic and may sometimes be important are:

(1) infinite population size,
(2) no selection,
(3) random mating (Hardy-Weinberg equilibrium),
(4) linkage equilibrium,
(5) no linkage.

All or part of these assumptions have been made in recent works on the expectations of the covariances between relatives.

The present study was undertaken to study the importance of the assumptions of infinite population size, no selection, and no linkage on some common procedures used to predict genetic improvement. Of particular interest was the effect upon the prediction of improvement

and how these effects differed in two sizes of populations with two different linkage relationships, two intensities of selection, and two levels of environmental variation.

REVIEW OF LITERATURE

This review concerns the development of the theory of predicting genetic improvement, since the object of the work is to study the consequences of assumptions that have necessarily been made. A comprehensive review of the studies which used simulated genetic populations was made recently by Gill (1963).

Formal consideration of predicting change was described at least as long ago as the definition of the simple linear regression coefficient b_{yx} by Gauss (1806). In general, b_{yx} expresses the expected change in an unobserved or dependent variable Y for each unit change in the observable or independent variable X. If y and x are taken as $Y - \bar{Y}$ and $X - \bar{X}$ respectively, the value of b_{yx} is chosen to minimize the sum of squared deviations of the dependent variable Y about the straight line extending over the range of X. A useful relationship is that $b_{yx} = r_{yx} \frac{\sigma_y}{\sigma_x}$, where r_{yx} is the correlation coefficient of x and y (Bravais, 1846), and σ_y and σ_x are the standard deviations of the dependent and independent variables, respectively.

Using the general notion of prediction equations, genetic improvement may be predicted as $\Delta G = b_{GP}\Delta P$, where ΔP is the average superiority of the phenotypic values of individuals selected to be parents of the next generation and b_{GP} is the regression of genetic value on phenotypic value, provided that the relationship is linear over the range

of phenotypic values. The regression coefficient, $b_{GP} = \frac{Cov(GP)}{\sigma^2_P}$, cannot be estimated directly when the genotypes are unknown, and thus their genetic value (whatever the measure) is unknown.

The genetic likeness of relatives, measured as covariances, provides indirect methods of estimating Cov(GP). The likeness of parent and offspring was observed long before any use was made of it. Berge (1961) in a history of the development of animal breeding says, "The ancient Greeks and Romans had, by comparison with later periods, a relatively highly developed standard of breeding of domestic animals. Approved methods of breeding were based on recognition of the fact that the parents leave their mark on the offspring." Engeler (1936) found evidence of the quantification of lineal relationship as early as 1815 in the work of Krünitz. He reported a developing importance of such a concept in Germany about 1860 as Individual potenzlehre following the work of Weckherlin (1851). Weckherlin had found that the influence of the ram on fiber diameter decreased about one-half for each ancestral generation.

Galton's "Law of Ancestral Heredity" (1897) is a milestone in quantifying the relations between lineal relatives. Galton (1889) tentatively attempted to give a precise mathematical expression of the resemblance between an offspring and its ancestors. Galton (1897) tested this hypothesis on the inheritance of black spots in Basset hounds. He

was sufficiently satisfied with the result to formulate the "Law of Ancestral Heredity," which states, "The two parents contribute between them, on the average one-half or (0. 5) of the total heritage of the offspring; the four grandparents, one-quarter or $(0.5)^2$; the eight great-grandparents, one-eighth or $(0.5)^3$ and so on. Thus the sum of the ancestral contributions is expressed by the series $(0.5) + (0.5)^2 + (0.5)^3$, etc., which, being equal to 1, accounts for the whole heritage."

Pearson (1898) was unable to substantiate Galton's conclusions and later (1903, 1904a, 1904b) disavowed the Law of Ancestral Heredity as a biological law. In fact, Pearson (1903) said, "This purely statistical and legitimate conclusion was seized upon as a biological law, and all life, but for constant selection, was in a state of regression to some distant ancestor." He concludes that Galton had only suggested the geometric series 1/2, 1/4, 1/8 . . . as a <u>à priori</u> assumption of the regression coefficients in predicting from ancestral deviations the most probable deviation of an offspring from its own generation. Yule (1902) had pointed out that the error in Galton's "Law of Ancestral Heredity" resulted from neglecting intercorrelations between the ancestors. The concept that, taken one by one, the regression on either parent is 0. 5, on either grandparent $(0.5)^2$, etc., remains a legitimate conclusion. This is quite different from squaring all of these and adding them together without reference to their inter-correlations. In an attempt to

bring Mendelism into the biometrical structure, Pearson (1904a) concluded that numerical values deducible from such principles would be a very great advantage, but that they did not agree with observation. Yule (1907) showed that this lack of agreement could be the result of the assumption of complete dominance as a principle. If no dominance was assumed, the coefficient of one-half based on the theory of segregation might be quite reasonable.

The mathematical basis of covariation of relatives was investigated by Snow (1910), Brownlee (1910), Weinberg (1908a, 1908b, 1909) and Jennings (1916, 1917), and Robbins (1917, 1918a, 1918b) prior to the classic work of Fisher (1918). In the latter, the correlations between relatives were defined in the most complete manner up to that time. His work is still considered to be essentially correct, except for the treatment of linkage and epistasis. Pearson (1904a) had treated the simple case of two alleles with equal frequency, and Weinberg (1908a, 1908b, 1909) had examined an arbitrary number of alleles and some cases of partial dominance. Fisher (1918) not only obtained a complete solution for additive effects, but also included dominance and a special case of epistasis that he called "dual epistacy," or the interaction between pairs of loci. Wright (1921a), unaware of Fisher's earlier work, used the method of path coefficients and also obtained useful results for the case of additive effects, but failed at that time to include the

correlation between dominance deviations in the case of full sibs.

Malécot (1948) expressed the general notions of Wright and Fisher in terms of probability. He showed that the covariance between any two individuals under random mating, X and Y, may be expressed as:

$$\mathrm{Cov}(X, Y) = \left(\frac{\phi + \phi'}{2}\right) \sigma^2_A + (\phi\ \phi')\sigma^2_D ,$$

where σ^2_A is the genic (additively genetic) variance, σ^2_D is the dominance variance, and ϕ and ϕ' are, respectively, the probabilities that genes from the sires (S_x and S_y) and the dams (D_x and D_y) are identical by descent.

Cockerham (1952, 1954) and Kempthorne (1954, 1955) extended the concepts of covariance among relatives to include epistasis. These works assume random mating, although parents may be inbred. Fisher (1918) had included the additive epistasis at two loci. However, for two alleles, Cockerham spelled out quite clearly the partitions of epistatic variance in terms of the well-known statistical technique of orthogonal polynomials. Kempthorne used the notion of derived models in factorial experiments (Kempthorne, 1952) to obtain more general results, which he related to the work of Malécot in terms of an arbitrary number of alleles. Following this generalization one may express the covariance

between any two individuals including epistasis as

$$\mathrm{Cov}(X, Y) = \sum_{\substack{r = 0, s = 0 \\ 1 \leq r + s \leq n}}^{n} \left(\frac{\phi + \phi'}{2}\right)^{r} (\phi\phi')^{s} \sigma^{2}_{A^{r}D^{s}} .$$

Cockerham (1956) expressed the effects of linkage in the expectations of the covariances between certain relatives in random mating populations. He concluded that the expectations of linear relationships, such as parent and offspring and grandparent and grandchild, were not affected by linkages. The inclusion of linkage has received considerable attention recently by Jones (1960), Schnell (1961, 1963), and Van Aarde (1963). The treatment has been extended to allow for an arbitrary number of loci, each with an arbitrary number of alleles, arbitrary dominance and epistasis, and any linkage relationships which are independent of gene effects. Schnell (1963) and Van Aarde (1963) have shown that only the parent-offspring relationship is unaffected by linkage rather than the more general class of relationships given by Cockerham (1956). Van Aarde (1963) and Schnell (1963) made the following assumptions in dealing with linkage:

(1) The populations considered are assumed to be infinte, random mating and at equilibrium.

(2) There is no selection, mutation, migration or other causes of changes in gene frequency.

(3) The population is in linkage equilibrium.

(4) There are no positon effects on genotypic values.

(5) Gametic ratios are independent of gene effects or environment.

Robinson and Comstock (1955) included the effects of linkage disequilibrium in finding the expectations of the male and female components of variance in the analysis of the offspring of bi-parental crosses of $\underline{n}$ females mated to each of $\underline{m}$ males with $\underline{o}$ offspring resulting from each mating (Design I, Robinson et al., 1949).

Other assumptions made by Van Aarde (1963) and Schnell (1963) are appropriate here. The following additional assumptions must be made:

(1) no epistasis

(2) no sex linkage

(3) no maternal effects

(4) only two alleles per locus

The progeny variance arising from genetic differences among female parents mated to the same male is σ^2_f and among male parents is σ^2_m. The expectations were shown to be

$$\sigma^2_m = 1/4\,\sigma^2_g + \sum_{ij}(pt - rs)_{ij}[1 + (1 - 2q_i)a_i][1+(1 - 2q_j)a_j]\,u_i u_j$$

$$\sigma^2_f = \sigma^2_m + 1/4\,\sigma^2_d + 2\sum_{ij}(pt - rs)^2_{ij}\,a_i u_i a_j u_j$$

where

σ^2_g = the additively genetic variance,

σ^2_d = the dominance variance,

u_i = one-half the distance between the effects of the homozygous genotypes at the i th locus,

$a_i u_i$ = the deviation of the effect of the heterozygous genotype from the means of the effects of the two homozygous genotypes at the i th locus,

q_i = the frequency of the favorable allele,

p = frequency of B_iB_j gametes, i. e., with the favorable allele at both the i th and the j th locus,

r = frequency of B_ib_j gametes,

s = frequency of b_iB_j gametes,

t = frequency of b_ib_j gametes.

Finite population size, selection and/or assortative mating generate linkage disequilibirum and departures from Hardy-Weinberg equilibrium. Such conditions make it difficult, if not impossible, to examine such expectations.

A review of the development of theories of predicting genetic improvement and the theory of the genetic expectations of the covariances between relatives must leave the reviewer concerned about the assumptions involved in such theoretical development. This concern may be relieved by some knowledge of the relative importance of such assumptions.

SIMULATION AND EXPERIMENTAL PROCEDURE

The intent of this study was to assess the relative importance of some of the assumptions made in developing the genetic expectations of the covariances between relatives and using this knowledge to predict genetic improvement. Simulation has been suggested as a useful tool for such investigation. Fraser (1960) suggested that one of the uses of such numerical methods is to examine methodically the importance of factors influencing the effectiveness of selection. Martin and Cockerham (1960) came to view the method as one of detection and suggestion. The present study may stimulate further theoretical development. Robertson (1960) suggested that simulation was one of the most effective ways of approaching the problem of joint effects of selection and linkage.

The study of infinite populations is impossible in practice, and extensive study of finite populations seems nearly impossible from a mathematical point of view. Simulation is not the solution to the study of infinite populations. Even large populations, in the computer simulation sense, will not begin to approach the infinite population problem. However, one may investigate different sizes of populations. Some conclusions may then be drawn about different finite populations and the direction of changes noted as the size of the population is increased. Thus simulation seems particularly appropriate to investigate the

importance of selection and linkage and may provide valuable insight into the importance of finite population size. However, genetic drift always remains a prominent and generally unknown aspect of such investigations.

A 2^3 factorial plan was chosen for this study. Such a plan enables one to compare the effects and interactions among three factors at two levels. The variable factors and levels were:

(1) Parent population size. Parent population sizes studied were eight and sixty-four males each mated to four females to obtain 256 and 2048 offspring. Each mating was allowed to produce four male and four female offspring. Crow (1954) has shown that when each mating produces the same number of offspring, the genetic drift is one-half of that expected when the number of offspring is distributed as Poisson.

(2) Linkage intensity. One autosome was simulated with recombination fractions of 0.50 and 0.01 between adjacent loci. An intensity of 0.50 is functionally the same as different autosomes.

(3) Selection intensity. The selection intensities studied were:

(a) selecting the best 1/16 of the male offspring and the best 1/4 of the females,

(b) selecting the best 1/8 of the males and practicing no selection on the females.

Two levels of environmental variance were also considered for

the 2^3 factorial plan. One level was to include no environmental variance and the second level was to include an amount equal to the expected initial genetic variance (20).

Levels of each factor were chosen with some thought of reality in mind, but limited by the available facilities. Population size may be considered in terms of either parent or offspring population. If a specified fraction of each sex is to be selected, the size of one population determines the other. In this study, population size was defined in terms of the number of parents since they determine the genetic material to be available and the amount of inbreeding to be expected in a finite population mating at random. The population of 2048 offspring was as large as was compatible with the computer. Eight times as many small populations were studied to make the total number of offspring studied comparable. The results from small populations were pooled in sets of eight before any analysis. A special problem was introduced by using different selection intensities while maintaining the parent and offspring populations at a constant size. The less intense selection from the same total number of offspring provides more selected parents than are needed in the next generation. This problem was avoided by reducing the size of the offspring populations from which selections were made. The less intense selection of males was accomplished by making selections from an offspring population consisting of only the first four offspring of each

mating. The females were selected at random from this reduced offspring population.

The effects of linkage on selection progress were an objective of simulation studies by Fraser (1957b), Martin and Cockerham (1960), Baker and Comstock (1961), and Gill (1963). They did not reach full agreement concerning the importance of linkage intensity as a force upon genetic progress. The lack of agreement may be, to some extent, the result of differences in simulation. This disagreement suggested careful consideration of the choice of linkage intensities. Fraser summarized the results of his simulation of genetic systems in 1962. He (1957b) made two replicate runs of populations with either four or fifty parents producing twenty and four offspring per mating, respectively, at each of five linkage intensities. Complete dominance was simulated on two autosomes of three loci each with two alleles per locus. Rate of advance was clearly correlated with the tightness of linkage. Simulation of only six loci with complete dominance makes the comparison of the results difficult except with regard to part of the work of Martin and Cockerham (1960). They simulated five and twenty loci with two alleles per locus on one autosome with either complete dominance or additive gene action. Free recombination and recombination of (0.01), between adjacent loci, were simulated. Initial populations were generated in linkage equilibrium. With five loci, the tight linkage slowed progress

of the genotypic mean only when no environmental variation was included with the additive model. In populations with twenty loci, tight linkage slowed progress in all cases. A few runs, including linkage intensities of 0. 30 and 0. 01 for the additive model, showed that progress was hindered by increasing tightness of linkage. Baker and Comstock (1961) simulated thirty-five loci with two alleles per locus on one autosome with complete dominance. Linkage intensity of (0. 01) did not slow the progress of the genotypic mean. Gill (1963) also found little effect of linkage. Five loci were simulated on each of eight autosomes with linkage intensities of 0. 005, 0. 05, 0. 20, and 0. 50 between adjacent loci. These latter results are in some conflict with the twenty loci results of Martin and Cockerham.

In all of these studies, linkage intensity was related to the probability of crossing-over between adjacent loci on the same chromosome. Intensity may also be defined in terms of the average relationship between $\frac{n(n-1)}{2}$ pairs of loci. The average relationships computed in this manner are shown in Table 1 for the previously mentioned studies, the present study, and the work of Qureshi (1963). The probability of recombination is computed for all possible pairs of loci by $r_{a,b} = 1/2[1 - (1 - 2r)^{b-a}]$ where $\underline{a}$ and $\underline{b}$ are linearly ordered loci numbered consecutively on a chromosome and $\underline{r}$ is the linkage intensity between adjacent loci. All relationships between loci on different

chromosomes are assumed to be 0.50. Calculating the average probability of recombination for loci on the same chromosome was simplified by using the following form:

$$\bar{r}_i = 1/2 \left[1 - \frac{1 - 2r}{n_i r} + \frac{(1 - 2r)^2}{2n_i(n_i - 1)r^2} \left\{ 1 - (1 - 2r)^{n_i - 1} \right\} \right] ,$$

where n_i is the number of loci on each chromosome. The average probability of recombination ($\bar{r}$) is then:

$$\bar{r} = .50 + \sum_i \frac{n_i(n_i - 1)}{n(n - 1)} \left[\bar{r}_i - .50 \right]$$

The comparison of Martin and Cockerham's results with twenty loci with those of Baker and Comstock and Gill, on the basis of $\bar{r}$ in Table 1, results in somewhat less disagreement concerning the effects of linkage intensity. A tight linkage intensity of 0.01 between adjacent loci on a single autosome was chosen in this study as likely to be intense enough to cause some observable differences and yet not be particularly unrealistic.

The details of simulation vary widely depending upon the computer to be used. No attempt will be made to detail this simulation. However, the genetic processes to be simulated and the assumptions to be made should be specified. The genetic system will be described in terms of the processes which were simulated and certain aspects which were not

simulated, although possibly important.

Dioecious diploid populations in which a quantitative trait is expressed equally in each sex were simulated. Regular diploid segregation and recombination was assumed for gamete formation. Zygotes were formed and the genotype was expressed as a genetic value for a complete additive mode of gene action. Where environmental effects were desired, a phenotypic value was expressed as the sum of the genetic value and an environmental component. Some specific simplification was achieved by assuming no mutation and no differential viability or reproductive rate (no natural selection).

Most quantitative traits are assumed to be affected by many loci. In practice one simulates as many loci as may be readily handled by the available computer. In the present study, the 40 binary "bit" structure of one computer "word" of memory was most adapted to 40 loci with two alleles per locus. This limitation makes the magnitude of individual gene effects large relative to the total variability. All loci were assumed to have equal effects. Unequal effects would cause the genes with large effects to be fixed more quickly. This would further decrease the effective number of loci. Equal recombination frequencies were assumed for adjacent loci because of the many ways inequality might be assigned. The use of non-overlapping generations also avoided a problem of specifying one of many possibilities. Barker (1958) suggests

that overlapping generations have little effect on most results. Similarly, Haldane (1926) found little difference in expressions of progress from selection whether generations overlapped or not.

Initial populations were produced by uniting specially constructed gametes. These gametes were generated as random binary numbers. Effectively, one of two alleles was randomly assigned to each of the 40 loci. The association between loci, in coupling or repulsion phase, should have been essentially random with gene frequencies about one-half at each locus. Initial disequilibrium should have been minimized. These initial populations are clearly different from those created by Gill (1963). Gill forced each and every locus to be heterozygous, and gene frequencies were then exactly one-half. One-half of the initial population was considered to be male and the other half to be female. Parents of the next generation were selected from these populations. In this study, a particular mating scheme was desired for the analysis of half-sib and full-sib familes. Each of the s selected sires was mated to four selected females to produce four male and four female offspring from each female parent. Matings were made at random, except that each female was mated to only one male. This was essentially a scheme of sampling without replacement. The totality of offspring form s half-sib families and four full-sib familes within each half-sib family. Segregation, recombination, and fertilization were simulated by the

random-mask method developed by Schweppe and Bohidar and used by Bohidar (1960) and Gill (1963). This method is particularly adaptable to a binary computer with the capability of logical arithmetic. In such a case, it is faster than the random transform of a vector of recombination frequencies used by Fraser (1957a), or the random-walk method suggested by Fraser (1960). The details of the random-mask method were outlined by Gill (1963).

The initial output from the computer consisted of means and components of variance and covariance. Sixteen replications were run for the 2^3 factorial with each of the two levels of environmental variance. A total of 128 small populations was simulated. Means and components of variance and covariance were pooled in sets of eight to make the sixteen replications of the small populations. Since this study was concerned with the prediction of genetic improvement, these results were combined into other variables for analysis. These final variables are ratios of predicted genetic improvement to actual improvement. Five common methods of predicting genetic improvement were considered:

(1) Correlation between full sibs (FS),

(2) Correlation between paternal half-sibs (PHS),

(3) Regression of offspring on mid-parent (MPO),

(4) Regression of offspring on dam (DO),

(5) Intra-sire regression of offspring on dam (DOS).

The computation is as follows:

$$(1)\quad FS = \frac{2(S + D)(\text{Reach})}{S + D + W} \div \Delta G$$

$$(2)\quad PHS = \frac{4\,S\,(\text{Reach})}{S + D + W} \div \Delta G$$

$$(3)\quad MPO = \frac{\text{Cov}(\overline{MPO})(\text{Reach})}{V(\overline{MP})} \div \Delta G$$

$$(4)\quad DO = \frac{2\,\text{Cov}(DO)(\text{Reach})}{V(D)} \div \Delta G$$

$$(5)\quad DOS = \frac{2\,\text{Cov}(DO/S)(\text{Reach})}{V(D/S)} \div \Delta G$$

where

S = the sire component of variance,

D = the dam component of variance,

W = the within full-sib family component of variance,

$\text{Cov}(\overline{MPO})$ = the component of covariance between offspring and the average of the parents,

$\text{Cov}(DO/S)$ = the component of covariance between offspring and their dams which were mated to the same sire,

$\text{Cov}(DO)$ = the component of covariance between offspring and dam,

$V(\overline{MP})$ = the component of variance among mid-parents

$V(D/S)$ = the component of variance among dams mated to the same sire,

$V(D)$ = the component of variance among all dams,

Reach = the average difference between selected males and females and the offspring populations from which they were selected,

ΔG = the actual genetic change in the genotypic mean in successive offspring generations.

DISCUSSION OF RESULTS

Prediction of Response to Selection

The primary aim of the study was to compare five frequently used methods of predicting genetic improvement when some of the assumptions underlying the methods were not satisfied. The importance and the relative importance of assumptions of no selection, no linkage and infinite population size were investigated by predicting genetic improvement and comparing the accuracy of the prediction relative to the observed improvement in simulated populations. Sixteen replications of two intensities of selection, two linkage intensities and two population sizes were studied in a 2^3 factorial plan. Results were obtained with and without environmental effects. The means of sixteen replications of the ratios of predicted to achieved genetic improvement are summarized in Tables 2 - 13. Tables 2 - 7 are results of using the regression of offspring on mid-parent (MPO), the regression of offspring on dam (DO), and the intra-sire regression of offspring on dam (DOS). Tables 8 - 13 are the results of using the correlation between full-sibs (FS), the correlation between paternal half-sibs (PHS) and their adjusted estimates (FSA and PHSA). The adjusted estimates have been adjusted for the reduced variability among selected parents. Tables 2 - 4, 8 - 10 are the results of including no environmental variance and Tables

5 - 7, 11 - 13 are the results of including environmental variance equal to the initial genetic variance. In the tables the first digit identifies selection (0 - intense, 1 - moderate). The second digit identifies population size (0 - large, 1 - small). The third digit identifies linkage (0 - 0.01, 1 - 0.50). The use of (.) denotes averages over that factor. For example, the means of the two levels of selection are (0.. and 1..).

The results of analyses of variance are presented in Tables 14 - 15 for no environmental variance and Table 16 - 17 for results including environmental variance. The nature of the results makes it convenient to consider the five methods of predicting genetic improvement as two groups. Methods DOS (using the regression of offspring on dam mated to the same sire), DO (using the regression of offspring on dam) and MPO (using the regression of offspring on their mid-parental average) will be considered collectively as "regression methods". Methods PHS (using the correlation between paternal half sibs) and FS (using the correlation between full sibs) will be grouped as "variance component methods". The discussion will also be divided according to the absence or presence of environmental variance.

"Regression Methods" - No Environmental Variance

Two general questions are to be considered. The first concerns the over-all accuracy of predicting improvement. The second is whether

there are significant differences in accuracy in different populations as intensity of selection, linkage intensity, and population size were varied. The answer to the question of over-all accuracy is reasonably clear when no environmental variance was included (Table 2). The "regression methods" adequately predict genetic improvement when averaged over the variable factors and the period of five generations. That is, there is no evidence that the averages of the ratio variables differ from unity. Ratios equal to 1.0 would be the result of perfect prediction of actual genetic improvement. Differences in accuracy may be assessed in the analysis of variance in Table 14. Only one statistically significant difference was found. Reference to Table 3 shows that the statistically significant ($P < .05$) interaction between levels of selection and generations for variable MPO should probably be of no concern. The differences were quite small and did not exhibit consistent trend in succeeding generations.

"Regression Methods" --Environmental Variance

When environmental variance was included, the "regression methods" may be considered to be generally accurate (Table 5). However, results were much more variable than in the absence of environmental variance. The analyses of the "regression method" variables in Table 15 show clearcut interactions between selection and generations

for all three methods and an interaction between selection and linkage for DO and DOS. Both interactions may be summarized by noting in Table 7 that, for intense selection and tight linkage, the "regression methods" overpredicted improvement somewhat in early generations and underpredicted considerably in the last two generations.

"Regression Methods" - A Discussion of the Relation to Theory

In this section consideration will be given to certain theoretical results which relate to using the "regression methods" to predict genetic improvement. Some attention will be given to the theoretical results of selection, random errors of measurement, non-normality, non-linearity, and their possible effects on the present study.

The literature of statistical genetics contains numerous, almost offhand, references to the statistical concept that the regression coefficient is not biased by selection of the independent variable. The estimation of regressions of offspring on parent treats the parent trait as the independent variable. Parents are almost certain to have been selected in some manner. It is well-known that if the regression is really linear, the estimate of the regression coefficient is not biased by selection of the independent variable. It is, then, an unbiased estimate as a description of the population from which the selected independent variables came. However, if selection was

practiced on some other variable, this simple result need not hold true. If selection was on some third or fourth variable, which is correlated with the dependent variable, then the simple regression coefficient will be biased. In this study, the simplest conditions hold true for considering the effects of selection on an independent variable. Selection was only on the independent variable, namely the parental phenotype. Thus, the estimates of the simple linear regression coefficient (offspring phenotypic value regressed on parental phenotypic value) would be expected to be unbiased estimates of the regression in the unselected population. The regression of offspring on one parent is used as an estimate of $h^2/2$, and the regression on the mid-parent is used as an estimate of h^2. Heritability (h^2) will be defined as a ratio of the genic variance to the total phenotypic variance. This is heritability in the narrow sense as carefully detailed by Lush (1961). The genic variance, also called the additively genetic variance, is the variance which can be attributed to the average effects of all genes as they exist in the population. The distinction between heritability in the narrow or broad sense (Lush, 1961) is not particularly important in this study because here all of the genetic variance is genic.

Shewhart (1926) and Winsor (1946) gave particularly lucid consideration to the effects of random errors of measurement on the estimation of the simple linear regression coefficient. Their results show that considering the regression of the genic value of offspring on the

genic value of one parent leads to the estimate of $h^2/2$ when all the other variance among offspring and parents is considered to be due to random errors of measurement. The non-genic variance included in this study may be considered in this manner.

Cochran (1951) mentioned non-normality as a remaining problem in the general framework of predicting genetic improvement. As a resume of the problem of non-normality it would be difficult to improve upon the following quotation from Cochran's paper. His use of $\underline{y}$ is comparable to the measure in the offspring generation and $\underline{x}$ to the measure of the parent.

> In view of the widespread assumption of normality in applications, an investigation of the consequences of this assumption in nonnormal populations would also be worthwhile. In general, a linear index will not be the best index, and predictions of the expected gain in y, based on normal theory, are likely to be in error. Unfortunately it cannot be taken for granted that a moderate departure from normality will have little effect. This may be so if selection is not intense and y has only a small correlation with the x's, so that progress is slow. But in intense selection the gains depend primarily on the shapes of the tails of frequency distributions. As is well known, a frequency curve which looks quite similar to the normal curve may differ greatly in its tail. A combination of theoretical investigations with sampling experiments on natural populations is suggested.

Some selection and non-normality is almost certain in any population. Errors of measurement which lead to less than perfect heritability are the general rule. Non-linearity, in the sense of dominance or epistasis, is likely in reality. The results of this study should not have been affected by this sort of non-linearity because gene effects were

entirely additive here. There also was no intentional correlation between genetic and non-genetic effects. The presence of these possible sources of theoretical failure and possible interaction among them leaves room to doubt that the regression of offspring phenotypic value on parental phenotypic value is appropriate in all cases.

Let us now consider those factors which might have influenced the accuracy of the "regression methods" (MPO, DO, and DOS) in predicting genetic improvement in this study. Selection was designed into this study. It may be well to be reminded that selection was always more intense among sires. The mild level of selection actually involved saving the best one-eighth of the males, but no selection of dams. Thus, for moderate selection, the effects of selection on DO and DOS are indirect through the selection of sires. There were no statistically significant direct effects of selection. However, the interaction of selection and linkage and selections and generations makes the interpretation of the direct effects of selection difficult.

Non-normality might have been pronounced in part of this study. The genetic values of initial populations were multinomially distributed. Phenotypic values, after including environmental effects which were approximately normally distributed, should have been very nearly normally distributed. When no environmental effects were included, the phenotypic values are the genetic values, but the multinomial

distribution of genetic values over 40 loci should be approximately normal. Selection of parents skews the distribution of genetic values in the offspring population. When environmental effects are included, the phenotypic values in the offspring population are somewhat less skewed by such selection. Tight linkage tends to increase the skewness. Successive generations of selection would increase these effects, particularly with tight linkage.

If the previous paragraph is essentially correct in describing the development of non-normality, it may explain the significant interactions found in using the "regression methods" to predict genetic improvement. Non-normality should be most distinct when selection is intense and linkage is tight and should increase in later generations. Such a development of non-normality follows the observed pattern of the interactions of selection and linkage and selection and generations when environmental variance was included. However, the picture may not be as clear as one might conclude at this point. Selection intensity, which may be most meaningfully considered in terms of the genotype, may be altered by varying either the fraction selected or the amount of environmental variance. However, the results may be different. The distribution of genotypes is probably distorted more severely when there is no environmental variance. If such were true, then the effects should have been most pronounced in populations where no environmental

variance was included. There was no indication of interactions between selection and linkage when no environmental variance was included.

Perhaps the most likely explanation of the significant interactions found in using the "regression methods" to predict genetic improvement may be traced to the use of regressions rather than correlations as estimates of heritability. The regressions are useful estimates of heritability only when the assumption may be made that the standard deviations of the unselected parent population and the offspring population are the same. Then the regression is equivalent to the correlation. That is $b_{yx} = r_{yx}\frac{\sigma_y}{\sigma_x}$, and $b_{yx} = r_{yx}$ only if $\sigma_y = \sigma_x$. Selection and tight linkage tend to reduce the standard deviation in the offspring population (σ_y) in relation to the unselected parent population (σ_x). The regression is thus less than the correlation and heritability is underestimated. Successive generations of selection would increase these effects. Underestimates of heritability lead to ratios less than 1.0 for predicted to achieved genetic improvement. Table 7 shows that such was the case when selection was intense and linkage was tight (0.01). Table 7 summarizes the results when environmental variance was included. No comparable result was shown when no environmental variance was included (Table 4).

"Variance Component Methods"- A Discussion of the Relation to Theory

The "variance component methods" (PHS and FS) are certainly not

accurate methods of predicting genetic improvement when the parents have been selected. The expected result is obvious in Tables 8 - 13. Since much of the inaccuracy was anticipated in advance, this aspect will be considered before a discussion of the results and their relation to this theory.

The selection practiced in this study reduces the variability among sires and dams. The effects of such selection received consideration by Reeve (1953). He investigated in detail the theoretical effects of selection which increases the variability among selected sires and/or dams and the effects of intentional assortative mating. However, he noted that the general approach is equally applicable to selection which reduces variability among parents. The present observed results will be compared with the results shown by Reeve.

Reeve (1953) developed methods of adjusting estimates of heritability for the effects of selection and assortative mating. He, very specifically, considered heritability in the narrow sense. He handled a special case of full-sib families resulting from mating one male to a single female. For this study, his work was developed somewhat further to include full-sib families and the paternal half-sib families which result when each male is mated to more than one female. This development is included in Appendix A. The correlations between paternal half sibs (r_{PHS}) and between full sibs (r_{FS}) have been expressed in

terms of selection and the true heritability. The ratio of genic variance to phenotypic variance in the unselected parental generation will be defined as true heritability (h^2). Figure 1 is a path diagram showing the correlation between paternal half sibs. The effects of selection have been defined as the values of K and K', where the variance among selected sires is a fraction (1 + K) of that among members of the population from which the parents were selected and the variance among selected dams is a fraction (1 + K'). Figure 2 is a path diagram showing the correlation between full sibs. In Appendix A a measure of assortative mating is included in the development. This quantity, M, is taken as a measure of the degree of phenotypic assortative mating as was done by Reeve (1953). It is the phenotypic correlation between mates and the r_{pp} of Wright's notation, rather than his m(Wright, 1921a). There was no intentional assortative mating in this study. However, some phenotypic correlation between mates necessarily occurs because of the finite number of matings in a particular population and generation. There should be as much positive as negative assortative mating, and M may be reasonably considered to fluctuate randomly around zero. In this study M was actually calculated. The overall average was .00026 with a range from - 0.20 to .18.

The usual computed estimates of heritability ($4r_{PHS}$ and $2r_{FS}$) may be expressed as follows:

$$a = 4r_{PHS} = h^2\left[\frac{1 + Kh^2}{1 + \bar{K}h^4/2}\right],$$

and

$$b = 2r_{FS} = h^2\left[\frac{1 + \bar{K}h^2}{1 + \bar{K}h^4/2}\right]$$

The average of K and K' is expressed as $\bar{K}$. These expressions can be solved as quadratic equations in h^2. The solutions are:

$$h_a^2 = \frac{-1 \pm \sqrt{1 + 4a(K - \bar{K}a/2)}}{2(K - \bar{K}a/2)}$$

and

$$h_b^2 = \frac{-1 \pm \sqrt{1 + 4b\bar{K}(1 - b/2)}}{2\bar{K}(1 - b/2)}$$

The quantity, h_a^2, is a paternal half sib estimate of heritability adjusted for the reduced variability among selected parents and h_b^2 is the adjusted full sib estimate. The adjusted full sib estimate of heritability is essentially that found by Reeve (1953). The one difference is the explicit inclusion of unequal selection among parents. This possibility was mentioned only casually by Reeve. In the following discussion the root which uses the positive square root part of the solutions will be referred to as the positive root, and the root using the negative square root part will be the negative root. Reeve was able to use the positive root as the desired solution for all cases when $\bar{K}$ was positive (selection increasing the variability among parents). The positive root is not

necessarily the proper solution when K and K' are negative, as in this study. For this study it was the negative root for all but one case when no environmental variance was included. The exception was h_b^2 when selection was moderate. The proper solution was the positive root for all cases in which environmental variance was included. The choice of solutions may be clarified by referring to Figures 4 and 5. When the adjusted estimate of heritability (h_a^2 or h_b^2) is expected to be near 1.0, as when no environmental variance is included, the estimate would logically be taken from the curve near that point. This part of the curve is plotted by the negative root solutions for all cases except h_b^2 and moderate selection. The negative root solution results in nonsense estimates for h_b^2 when selection was moderate. Adjusted estimates of heritability ≤ 0.5 are taken from the part of the curve plotted by the positive root solutions.

When selection is such that the variability of parents is reduced, K and K' are negative, and a complication is introduced which did not concern Reeve. There is no "real number" solution if $4a(K - 1/2\bar{K}a) \leq 1$ or $4b(K1 - 1/2b) \leq 1$. This can and did happen in this study where $\underline{a}$ and $\underline{b}$ are subject to sampling variation. In such cases the square root expressions were taken to be zero.

The ratios of predicted to observed genetic improvement using adjusted estimates of heritability, h_a^2 and h_b^2, are shown in Tables 8-13.

The adjusted variables are identified as PHSA and FSA in contrast to the unadjusted ratios PHS and FS.

"Variance Component Methods" - No Environmental Variance

It has been previously mentioned that the "variance component methods" were not expected to be accurate when the parents were selected. Selection in this study reduced the variability among parents and thus the components of variance for sires (S) and dams mated to the same sire (D). Therefore, the sire component of variance and the dam component of variance are not estimates of one-fourth of the genic variance in the unselected parent population. This is true even though there is no dominance variance or epistatic variance. We still wish to consider the differences in accuracy in populations which differ with respect to the intensity of selection practiced, the intensity of linkage, population size, and their interactions. The results, when no environmental effects were included, are summarized in Tables 8 - 10. The analyses of these results are presented in Table 16.

The techniques of adjusting for the effects of selection were discussed in the previous section. This adjustment is in terms of the reduction in variability among sires ($1 + K$) and the average reduction in variability among both parents ($1 + \bar{K}$). Saving a constant fraction of a rather finite population does not necessarily mean a constant reduction

in the variability among selected parents. For example, selecting the best one-sixteenth of the potential sires resulted in widely different reductions in variability (variability among selected sires expressed as the fraction 1 + K of the variability in the unselected population). The fractions (1 + K) in different replications and different combinations of population size and linkage intensity sometimes differed in size by as much as three times. The observed results, in terms of estimates of heritability and hence prediction of genetic improvement, were more a function of the reduction in parental variability than a function of the fraction selected. The two levels of selection were defined in terms of the fraction of each sex to be selected. When defined in this way the effects of selection and interactions with selection may be confounded with other factors such as population size and linkage intensity. Particular combinations of selection intensity and linkage intensity, and population size may also affect the reduction in variability among the selected parents.

The extent of the complication resulting from the particular definition of selection intensity was investigated by analyzing the actual reduction in variability among parents. Values of $(1 + K)$ and $(1 + \bar{K})$ are presented in Tables 18 - 20. Analyses of variance are given in Table 24. Since $(1 + K)$ and $(1 + \bar{K})$ are actually fractions, the analyses were completed after an arcsin transformation. The analyses show

rather conclusively that the intensity of selection (defined in terms of the fraction selected) was not the only important factor. A more detailed discussion will be undertaken later as the similarity is shown between these analyses and the analysis of PHS. At this point it may be sufficient to note that the effect of linkage and the interaction of selection and linkage were statistically significant when no environmental effects were included.

Most of the results obtained by using the "variance component methods" can be considered appropriately only after correcting for the known reduction in the variability among the selected parents. Two exceptions will be made. First, it will be worthwhile to note that there was considerable reduction in the PHS variables between the first and second generation and again between the second and succeeding generations. There was a similar sharp decrease for FS between the first and later generations. Second, it may at first appear odd that the PHS variables were generally lower for moderate selection than for intense selection. This is a peculiarity of the manner in which moderate selection was accomplished in this study. If the denominator of a fraction is decreased proportionately less than the numerator the size of the fraction is reduced. Thus, if the component parts of the denominator of the paternal half-sib estimate of heritability are decreased proportionately less than the sire component of variance the estimate will be lower.

In the case of intense versus moderate selection, as practiced in this study, the size of the dam component of variance is decreased considerably by selecting the best one-fourth with intense selection in contrast to applying no selection among dams under the moderate selection scheme. The size of the sire component of variance, which is the numerator and part of the denominator, is decreased less distinctly by selecting one-eighth rather than one-sixteenth of the sires. The net result is that the denominator is decreased proportionately less than the numerator when selection is less intense and the estimate of heritability is lower. Circumstances such as this may occur when the intensity of selection is different for the sexes.

The reasons for considering the "variance component methods" after adjusting for the known reduction in the variability of parents (PHSA and FSA) should be clearer when one carefully considers the analysis of (1 + K). There are important similarities between the results of analysis of (1 + K, Table 21) and the results of the analysis of PHS (Table 16) where no environmental effects were included. The most important similarity is the interaction of selection and linkage. This interaction clearly resulted from the particular combination of moderate selection and tight linkage. In the case of (1 + K) the variability was reduced more than expected on the basis of the fractions selected in moderate selection. The same combination of selection

and linkage appeared to be the principal cause of the observed interaction between selection and linkage in the analyses of PHS. With the moderate selection, ratios of predicted to achieved genetic improvement were much lower than expected. The magnitude of the difference increased in succeeding generations for both (1 + K) and PHS.

The fundamental validity of the techniques developed to adjust for the effects of selection may be established by considering PHSA and FSA in Tables 8 - 10. The adjusted ratios of predicted to achieved genetic improvement did not differ significantly from 1. 0 in the initial generation. Adjustments made with the negative root result in ratios greater than 1. 0 when the unadjusted estimates of heritability were lower than was expected on the basis of the reduction in variability among the selected parents. Adjustments made with the positive root result in ratios less than 1. 0 when unadjusted estimates were low. In all but the initial generation, the variables (PHSA and FSA) were different from 1. 0. When the positive root was used (FSA with moderate selection) the ratios were less than 1. 0. When the negative root was used (all other cases of PHSA and FSA) the ratios were greater than 1. 0. The evidence indicates that unadjusted estimates of heritability were lower than was expected in all but the initial generation. The adjustment appears to be satisfactory in the initial generation. Failure in later generations may be related to an explanation of the effects

observed for selection intensity, linkage intensity, and particularly the interaction of selection and linkage. This is considered in a later section.

Since the adjustment for the reduction in variability among selected parents appears to be satisfactory in the first generation, the analyses of PHSA and FSA shown in Table 16 are for generations two to five only. The conclusions to be drawn concerning the analysis of FSA will be tempered by the knowledge that the adjustment for the effect of two levels of selection was confounded by the use of different roots. Intense selection was adjusted with the positive root and moderate selection with the negative root. When full-sib estimates of heritability are lower than can be accounted for by the reduction in the variability among parents, FSA is low for moderate selection and high for intense selection. The effect of selection intensity is magnified considerably as a result and, at best, is hazardous to interpret. The interaction of selection and linkage appears to be important even if the interpretation is tempered by this complication. The primary cause of the interaction appears to be the particular combination of intense selection and free recombination. This is most important! Levels of selection were the only statistically significant effect found in the analysis of PHSA. However, a careful consideration of PHSA means in Table 10 shows that the combination of intense selection and free recombination (0. 1) is

consistently the highest in each generation. This observation is additional evidence of the importance of this particular combination of selection and linkage.

Most of the results observed for PHSA and FSA can be traced to heritability estimates which are even lower than was expected to result from the reduced variability among parents. A detailed discussion of a possible cause and the particular effect of selection and linkage will be given after considering the results when environmental variance was included.

"Variance Component Methods" - Environmental Variance

The results found when environmental variance was included tended to be similar to those found where there was no environmental variance. The principal difference was that effects were not as clear cut, and results were generally more variable. The results are summarized in Tables 11 - 13. The analyses of these results are presented in Table 17.

These results will be considered largely on the basis of analyses of PHSA and FSA. However, the complication which resulted from defining levels of selection in terms of the fraction saved was not a serious problem when environmental variance was included. The values of $(1 + K)$ and $(1 + \bar{K})$ are presented in Tables 22 - 24. Analyses of

variance, after arcsin transformation, are given in Table 25. These analyses show that the intensity of selection and generations were the only important sources of variation. The differences in selection were intentional, and the differences in generations, for $(1 + \bar{K})$, did not seem to follow any particular trend.

One of the points considered to be important in the results for PHS and FS (no environmental variance) needs to be repeated here. There was a considerable reduction in the size of the PHS and FS variables from the initial generation to succeeding generations. There appears to be no comparable result when environmental variance was included. In actuality one would have expected an increase in successive generations. An increase would be expected because estimates of heritability are less severely reduced, by a given reduction in variability among selected parents, when true heritabilities are lower. (See Figure 3). The ratio of genic variance to phenotypic variance (true heritability) is decreased each generation as genic variance is decreased and the environmental variance is kept constant. Genic variance must decrease as selection increases gene frequencies from their initial point of about one-half. Genic variance is at a maximum when gene frequencies are at exactly one-half. The ratios of predicted to achieved genetic improvement (PHS and FS) would be expected to increase as the estimates of heritability were more nearly the value of true heritability. PHS and

FS did not increase toward 1.0 as expected, and we may conclude that these results are consistent with observations made when no environmental variance was included.

The results of the analyses of PHSA and FSA are not particularly different from the analyses of PHS and FS. This was to be expected when the analyses of $(1 + K)$ and $(1 + \bar{K})$ showed that the only significant differences were between selection intensities and among generations. However, to be consistent, consideration will again be given to PHSA and FSA in generations 2 - 5. The validity of the techniques developed to adjust for the effects of selection was not as clearly established when environmental variance was included. The method worked reasonably well for adjusting the full sib method (FSA) in the first generation. The adjustment did not work for the paternal half sib method (PHSA) when selection was mild. The average ratio of predicted to achieved genetic improvement for mild selection (1..) was clearly less than 1.0. There seems to be no simple explanation. Several populations had very small estimates of heritability. However, the results were not particularly more variable than other comparable results. The adjustment for the reduced variability among parents worked for all other cases in the first generation and broke down in later generations.

Consideration of all of the results reveals an overall picture of significant effects of selection, linkage, the interaction of selection and

linkage and some interactions with generations. A notable exception is the results for PHSA with environmental variance. These results are particularly disturbing because the consistency and trend from generation to generation was absent. The analysis showed no evidence of an average effect of selection. The difference between intensities of selection was statistically significant in generation 4, but opposite in sign to the differences in generations 2, 3 and 5. The observed interaction between selection and linkage was inconsistent with most other results. In this case the interaction resulted because the combination of mild selection and free recombination (1. 0) was rather consistently low. All other evidence of interaction between selection and linkage came largely from the combination of intense selection and tight linkage (0. 1). The highly significant effect of linkage was reversed from other results. The results for FSA were more consistent with the results when no environmental variance was included. The interaction of selection and linkage generally came from the combination of intense selection and free recombination.

"Variance Component Methods" - the Failure of the Adjustment

Two major results were shown in the study of the use of "variance component methods" to predict genetic improvement. First, the methods developed to adjust for the effects of reduced variability among

selected parents were effective for only the first generation. The general picture, particularly in later generations, was one of heritability estimates which were lower than was to be expected as a result of the reduced variability among selected parents. Second, there was consistent evidence of an interaction between the effects of selection and linkage. The interaction was largely the result of a particular combination which led to heritability estimates which were particularly low. What ever effects of selection or linkage were observed, they were generally associated with the interaction. This section develops a single explanation of both of these results.

The assumption will be made that these results had their origin in the estimates of heritability. There are at least two forms of evidence that this is a reasonable assumption. First, the heritability estimates (no environmental variance) were analyzed in the same manner as were the ratios of predicted to actual genetic improvement. These analyses were nearly identical to the analyses of the ratios. Second, an analysis of the achieved heritabilities (Table 26) detected no significant differences. Such an analysis is not conclusive, but is relevant. Ratios of predicted to actual genetic improvement may also be considered as a ratio of an estimate of heritability divided by achieved heritability. Achieved heritability is defined as actual genetic improvement divided by phenotypic reach. Unfortunately, both forms of

evidence are possible only in the absence of environmental variance. Such evidence is not available when environmental variance was included because actual heritability was changing as the average gene frequency was increased by selection. However, the available evidence indicates that the failure originated in the heritability estimates.

Recall that results from the "variance component methods" are most meaningful after adjustment for the effects of the reduced variability among selected parents. Let us also reemphasize the exact procedure used to adjust the estimates of heritability. In Appendix A we have shown how reduced variability among parents might be expected to affect the correlation between paternal half sibs (r_{PHS}) and the correlation between full sibs (r_{FS}). The usual estimates of heritability ($4r_{PHS}$ and $2r_{FS}$) may be expressed as follows:

$$a = 4r_{PHS} = \frac{[1 + \bar{K}h^2]\sigma^2\{H\}}{[1 + (1/2)\bar{K}h^4]\sigma^2\{P\}}$$

$$b = 2r_{FS} = \frac{[1 + \bar{K}h^2]\sigma^2\{H\}}{[1 + (1/2)\bar{K}h^4]\sigma^2\{P\}}$$

The variability remaining among selected sires is expressed as $(1 + K)$ and the average of the variability remaining among sires and dams as $(1 + \bar{K})$. The genic variance in the unselected parent population is symbolized as $\sigma^2\{H\}$ and the phenotypic variance as $\sigma^2\{P\}$. These expressions may be solved as quadratic equations in h^2. The solutions are:

$$h_a^2 = \frac{-1 \pm \sqrt{1 + 4a(K - \bar{K}a/2)}}{2(K - \bar{K}a/2)}$$

$$h_b^2 = \frac{-1 \pm \sqrt{1 + 4b\bar{K}(1 - b/2)}}{2\bar{K}(1 - b/2)}$$

The choice of a solution for a particular circumstance is critical. In the following discussion the solution which uses the positive square-root portion of the solutions will be referred to as the positive solution, and the solution using the negative square-root portion will be considered the negative solution. For this study the negative solution is appropriate for all but one case when no environmental variance was included ($h^2 = 1.0$). The exception is for full sibs when selection was mild. The logic of the choice of solutions may be seen in Figures 4 and 5. Furthermore, from these figures one may also observe the heritability (h^2) will be overestimated by low estimates of h_a^2 and h_b^2 when the negative solution is used and will be underestimated by low estimates of h_a^2 or h_b^2 when the positive solution is used. This is true regardless of the reason the estimates of h_a^2 and h_b^2 are low.

In the discussion of results in the two previous sections we have noted that when the ratios of predicted to actual genetic improvement differed from 1.0 it could generally be traced to heritability estimates which were too low. That is, genetic improvement was overpredicted when the adjustment was made using the negative solution and underpredicted when the adjustment was made using the positive solution.

The heritability estimates may be lower than expected in two ways. The numerator may be smaller than expected, or the denominator larger or both. Detailed consideration of the components of variance, which eventually make up the estimates of heritability, indicates that the estimates were low because the denominator was larger than was to be expected on the basis of the development shown in Appendix A. The evidence to be presented to support this conclusion is based completely upon results where no environmental variance was included, and we may assume that the true heritability is 1.0 in each and every case.

The development in Appendix A leads us to expect the sire component of variance (S_{n+1}) to be $(1/4)(1 + K)\sigma^2\{H_n\}$, where the variance among selected sires is defined to be the fraction $(1 + K)$ of the variance among members of the population from which they were chosen $\sigma^2\{H_n\}$. The dam component of variance (D_{n+1}) was expected to be $(1/4)(1+K')\sigma^2\{H_n\}$, where the variance among selected dams is the fraction $(1 + K')$ of the variance among members of the population from which they were chosen. The within full-sib familes component of variance (W_{n+1}) was expected to be $(1/2)\sigma^2\{H_n\}$ when there was no environmental variance. Tables 27 - 29 present means of ratios of actual to expected components of variance. There is no reason to believe that S or D were other than expected since the ratios are close to 1.0. However, there is every reason to believe that W was larger than

expected in all but the first generation. An increase in W makes the denominator of the heritability estimate larger than expected and lowers the estimate. The discrepancy in W was particularly large in populations which were intensely selected and were allowed to recombine freely. Analyses of generations 2 - 5 are presented in Table 30. The first generation was omitted since linkage disequilibrium could not have affected these results. The effects of selection, linkage, and their interaction were all highly significant ($P < .01$). The effects of selection and linkage are almost wholly the result of the interaction between selection and linkage.

It is clear that the variance within full-sib families (W_{n+1}) is larger than $(1/2)\sigma^2\{H_n\}$ except when n = 0, the initial population. Upper truncation selection, such as practiced in this study, favors repulsion gametes and would be expected to generate negative linkage disequilibrium or a negative correlation between the effects of different loci. Such disequilibrium makes the actual genic variance ($\sigma^2\{H\}$) less than might be considered as potentially available. To simplify several of the following equations, the summation over a single subscript $\Sigma_{i=0}^{n}$ will be written Σi and the double summation over two subscripts $\Sigma_{i=0}^{n}\Sigma_{j=0}^{n}$ for $i \neq j$ will be written Σij. The potential genic variance is $2\Sigma i p_i q_i \alpha_i^2$, where p_i and q_i are the frequencies of the two alleles and α_i is the average effect of a gene substitution at the ith locus. We can

represent the relationship between actual genic variance and the potential genic variance as

$$\sigma^2\{H\} = 2\Sigma ip_iq_i + 2\Sigma ijD_{ij} \qquad (1)$$

since $a_i = a_i = 1$ for all i and j and $2D_{ij}$ is the covariance (linkage disequilibrium) between the effects of loci i and j. The evidence in Table 27 makes it reasonable to accept

$$S_{n+1} = (1/4)(1 + K)\sigma^2\{H_n\}$$

and

$$D_{n+1} = (1/4)(1 + K')\sigma^2\{H_n\}$$

because the true heritability (h^2) is 1.0.

Then

$$\sigma^2\{H_{n+1}\} = S_{n+1} + D_{n+1} + W_{n+1} = (1/2)(1 + \bar{K})\sigma^2\{H_n\} + W_{n+1} \qquad (2)$$

Lush (1948) has shown that a fraction (the recombination fraction) of the disequilibrium between two loci is lost in a generation of random mating. We may then conclude

$$\sigma^2\{H_{n+1}\} = (1 + \bar{K}/2)\sigma^2\{H_n\} - 2\Sigma ijr_{ij}D_{ij} \qquad (3)$$

if we accept

$$\sigma^2\{H_n\} = (1 + \bar{K}h^4/2)\sigma^2\{H\}$$

as shown in Appendix A where linkage equilibrium must be assumed.

We can then equate (2) and (3) to find

$$W_{n+1} = (1/2)\sigma^2\{H_n\} - 2\Sigma ijr_{ij}D_{ij} . \qquad (4)$$

Since the quantity ($- 2\Sigma ijr_{ij}D_{ij}$) must almost certainly be positive for populations subjected to truncation selection, this is at least one explanation of why W_{n+1} was clearly larger than expected. It is an intuitively satisfactory explanation of the observed results, particularly for the interactions resulting from the combination of intense selection and free recombination. The disequilibrium (D_{ij}) should be largest as a result of intense selection, and free recombination ($r_{ij} = 0.50$) could make $\Sigma ijr_{ij}D_{ij}$ relatively large.

Any attempt to calculate $\Sigma ijr_{ij}D_{ij}$ would be very difficult, but from (1)

$$- 2\Sigma ijD_{ij} = 2\Sigma ip_iq_i - \sigma^2\{H\} . \qquad (5)$$

The potential genic variance ($2\Sigma ip_iq_i$) was calculated in eight of the sixteen replications, and $\sigma^2\{H\}$ was calculated in each replication. The results are summarized in Table 31 (no environmental variance) and Table 32 (environmental variance). We do not have a direct estimate of ($- 2\Sigma ijr_{ij}D_{ij}$) and it is this quantity which bears directly on W in (4). We can estimate this quantity if either the r_{ij} or the D_{ij} are the same for all $\underline{i}$ and $\underline{j}$. We know that $r_{ij} = 0.50$ for free recombination.

$- 2\Sigma ijr_{ij}D_{ij} = - 2\bar{r}\,\Sigma ijD_{ij}$ if all r_{ij} are the same, and

$- 2\Sigma ijr_{ij}D_{ij} = - 2D_{ij}\Sigma ijr_{ij} = - 2\bar{r}\Sigma ijD_{ij}$ if all D_{ij} are the same.

Then, from (5)

$$- 2\bar{r}\Sigma ijD_{ij} = \bar{r}[2\Sigma ip_iq_i - \sigma^2\{H\}]. \qquad (6)$$

The appropriate values of $\bar{r}$ are known to be .114 for the tight linkage (0.01 between adjacent loci) and .500 for free recombination. One can consider whether it is reasonable to assume

$$E(W_{n+1}) = 1/2\sigma^2\{H_n\} + \bar{r}[2\Sigma ip_iq_i - \sigma^2\{H_n\}]. \qquad (7)$$

which we get by substituting (6) into (4). Estimates of $\bar{r}(\bar{r}')$ were found to minimize

$$\sum_{n=1}^{6}\left[1 - \frac{W_{n+1}}{E(W_{n+1})}\right]^2$$

for each of the four combinations of selection and linkage.

Selection	Linkage	$\bar{r}$	$\bar{r}'$
intense	0.01	.114	.103
intense	0.50	.500	.460
moderate	0.01	.114	.620
moderate	0.50	.500	.472

Generation averages were used since Σip_iq_i was not calculated in every replication. The different population size results were kept distinct so

that twelve observations were available for each estimate.

Three of the four values of $\bar{r}'$ are reasonably close to the actual value of $\bar{r}$. An estimate such as that for moderate selection and tight linkage does not in anyway invalidate the other, more positive, results. Such an estimate could result if the D_{ij} were unequal and larger values of D_{ij} are for pairs of loci for which the recombination fraction was relatively large (loci near opposite ends of the chromosome). The estimates made from the results of free recombination ($\bar{r} = 0.50$) indicate that linkage disequilibrium is an adequate explanation for the behavior of the variance component $\underline{W}$ and much of the results of this study. Estimates of $\bar{r}(\bar{r}')$ near the actual value indicate that (7) is, at least operationally, reasonable. This does not prove the D_{ij} are the same or that linkage disequilibrium is the explanation of the results of this study.

Tables 31 and 32 and Figure 6 furnish additional evidence of the importance of linkage disequilibrium. The four combinations of selection and linkage are shown in Figure 6. Any differences in the results of different sizes of populations were relatively unimportant throughout this study. These results are presented as ratios in order to remove the effect of the total amount of variability present. Disequilibrium is important only as it affects the proportion of the potential variance which is actually available. There was remarkable consistency between

the results with and without environmental variance. The major difference was that the effects were more severe with no environmental variance. Certainly this would be expected. The increase continues through the fifth generation for intense selection and tight linkage and tends to level out at the second generation for free recombination. Moderate selection and tight linkage presents a very curious result in generations one and two. There was recovery in generation two and continued increase through generation five.

Eight replications (no environmental variance) were allowed to continue for two generations without further selection. These were the same replications in which the potential genic variance was calculated. The results are shown in generations six and seven of Table 31. The cessation of selection allowed $\sigma^2\{H\}$ to increase. There were large increases in the first generation of no selection and further average increase for all types of populations except with moderate selection, small population, and tight linkage (110). The increase in $\sigma^2\{H\}$ was generally much larger with free recombination.

There is sufficient evidence to conclude that both major results found in using the "variance component methods" can be attributed to the inability to account for the recovery of linkage disequilibrium. This meant that the denominators of the heritability estimates were larger than expected in all but the first generation. There was

essentially no disequilibrium in the initial population, and the methods employed to adjust for the reduced variability among selected parents were relatively satisfactory in the first generation. Intense selection generates more disequilibrium, and free recombination allows more of it to be recovered in the full-sib family component of variance (W). The result seemed to be an interaction between selection and linkage as the heritability estimates are reduced by the increase in W.

SUMMARY AND CONCLUSIONS

The complexity of any biological system precludes general theory for predicting genetic improvement. Present theory is necessarily restricted by the limitations of the assumptions which must be made. Simulation allows the investigation of the consequences of such limitations and may point the way to further fruitful developments in theory.

This study was undertaken to study the importance of the assumptions of infinite population size, no selection, and no linkage on some frequently used methods of predicting genetic improvement. The accuracy of the methods and differences in accuracy was considered in two sizes of populations with two intensities of selection, two linkage relationships, and two levels of environmental variation. A 2^3 factorial plan for each level of environmental variation enables one to compare the effects and interactions of selection, linkage, and population size. The parent population sizes were 8 and 64 males each mated to four females to obtain 256 and 2,048 offspring, respectively. The selection intensities were:

(1) selecting the best 1/16 of the male offspring and the best 1/4 of the females (intense),

(2) selecting the best 1/8 of the males and practicing no selection on the females (moderate).

The linkage relationships were:

(1) recombination of fraction of 0.01 between adjacent loci (tight),

(2) recombination fraction of 0. 50 (free).

The two levels of environmental variance were to include no environmental variance and to include an amount equal to the expected initial genetic variance (20).

Dioecious diploid populations in which a quantitative trait is expressed equally in each sex were simulated. Regular diploid segregation and recombination was assumed for gamete formation. Zygotes were formed and the genotype was expressed as a genetic value, assuming a completely additive mode of gene action. Where environmental effects were desired, a phenotypic value was expressed as the sum of the genetic value and an environmental component. It was assumed that there was no mutation and no differential viability or reproductive rate (no natural selection). Forty loci with two alleles per locus were simulated on a single autosome. Initial populations were produced by uniting specially constructed gametes. These gametes were generated as random 40 "bit" binary numbers. Effectively, one of two alternative alleles was randomly assigned to each of the 40 loci. Initial disequilibrium should have been minimized. Parents for the first offspring generation were selected from such an initial population. A particular mating scheme was desired for the analysis of half-sib and full-sib families. Each of the $\underline{s}$ selected sires was mated to four selected females to produce four male and four female offspring from each

female parent. Matings were at random, except that each female was mated to only one male in a scheme of sampling without replacement.

The initial output from the computer consisted of means and components of variance and covariance. Sixteen replications were run for the 2^3 factorial at each of the two levels of environmental variation. Means and components of variance and covariance were pooled from eight small population runs to make up one replication of the small populations. Ratios of predicted to actual genetic improvement were created from these initial results. Five frequently used methods of predicting genetic improvement were considered:

(1) Correlation between full-sibs (FS),

(2) Correlation between paternal half-sibs (PHS),

(3) Regression of offspring on mid-parent (MPO),

(4) Regression of offspring on dam (DO),

(5) Intra-sire regression of offspring on dam (DOS).

Methods (1) and (2) were also considered after adjustment for the reduced variability among selected parents and were identified as FSA and PHSA, respectively. Actual genetic improvement was taken to be the observed change in mean genetic value.

The results were considered separately as "variance component methods" (FS, PHS, FSA and PHSA) and "regression methods" (MPO, DO and DOS). The discussion was also divided according to the absence

or presence of environmental variation.

The "regression methods" were generally accurate means of predicting genetic improvement with and without environmental variation. As expected, the results were more variable when environmental variation was included. Analyses of variance showed statistically significant ($P < .01$) interactions between selection and generations for all three "regression methods" and an interaction between selection and linkage for DO and DOS when environmental affects were included. Both interactions seemed to result from overpredicted genetic improvement in early generations and considerable underprediction in the last two generations for the combination of intense selection and tight linkage. Several possibilities were considered as possible explanations of these results. Two of these possibilities seem more likely. Neither can explain why the interactions were not also detected when no environmental variance was included. Non-normality probably was most distinct when selection was intense and linkage was tight. However, it should be most distinct in the absence of environmental effects. The simplest explanation seems to be the use of regressions instead of correlations as estimates of heritability. The regressions are useful estimates of heritability only when it may reasonably be assumed that standard deviations of the unselected parent population and offspring population are the same so that the regression is equivalent to the correlation.

Selection and tight linkage reduced the standard deviation in the unselected offspring population in relation to the unselected parent population. The regression is less than the correlation, and heritability is therefore underestimated. Heritability was underestimated in the last two generations of intensely selected populations with tight linkage. A comparable result was not found when no environmental variance was included, and the overestimation in early generations may not be explained in this manner.

The "variance component methods" are not directly accurate methods of predicting genetic improvement when the parents have been selected. Adjustments for the reduced variability were developed from work by Reeve (1953). Selecting a constant fraction of a finite population does not mean a constant reduction in the variability among selected parents. Different intensities of selection would be expected to affect the reduction in variability. The actual reductions in variability measured as fractions ($1 + K$ and $1 + \bar{K}$) of the unselected parent population variance were analyzed to consider the possibility that the other factors included in this study might also affect the reduction in variability. Such analyses showed that there was an effect of linkage and interaction between selection and linkage. Analyses of the unadjusted ratios of predicted to achieved genetic improvement appeared to be very similar to these analyses and suggested that useful interpretations might be

made only after adjusting for the reduced variability among selected parents. The adjustment for the effects of reduced variability among parents appeared to be fundamentally valid. The adjusted ratios of predicted to achieved genetic improvement did not differ significantly from 1.0 in the initial generation. The failure in later generations appeared to be the result of unadjusted estimates of heritability which were lower than expected on the basis of the reduced variability among selected parents. After adjustment, the principal result of analyses of the two "variance component methods" with and without environmental variation was the indication of an interaction between linkage and selection. This interaction appeared to be the result of the particular combination of intense selection and free recombination. Such a general statement does not hold true for the paternal half-sib method (PHSA) when environmental variance was included. The adjustment for the reduced variability among selected parents failed in the first generation for those populations subjected to only moderate selection. The usual consistency and trend through generations was also absent here.

Linkage disequilibrium appeared to be one explanation of most of the results using "variance component methods" to predict genetic improvement. The within full-sib familes component of variance was larger than expected. Therefore, the denominator of the estimates of heritability were larger than expected and necessarily reduced the

estimates. The effect was particularly pronounced in populations which were intensely selected and allowed to recombine freely. Evidence was shown for the presence of considerable disequilibrium. The ratios of potential genic variance to actual genic variance generally increased in successive generations when linkage was tight. The effect seemed to level out at generation two with free recombination. When selection was stopped for two generations, the loss to disequilibrium began to be recovered. The recovery to actual genic variance was larger with free recombination than with tight linkage.

The results of this study indicate that regressions of offspring on parents may generally be used for relatively accurate prediction of genetic improvement in a population under selection. There were indications of inaccuracy when linkage was tight and selection was intense. A completely satisfactory explanation was not found. Estimates of heritability made from components of variance do not result in accurate predictions of genetic improvement in a selected population. Estimates of some of the components of variance are reduced when selection has reduced the variability among parents. However, such estimates may be adjusted for the effects of this reduced variability. This study emphasized that such adjusted estimates may be used for reasonably accurate prediction of genetic improvement in a population which is in linkage equilibrium. There was evidence that selection generated

disequilibrium in all of the simulated populations in this study. It was extreme in populations in which selection was intense and linkage was tight. In predicting genetic improvement, the effects appeared to be caused by the recovery of the disequilibrium. The recovery made the full-sib families component of variance larger than expected. Estimates of heritability were therefore lower than expected on the basis of the reduced variability among selected parents. The prediction of genetic improvement, after adjustment for the reduced variability among selected parents, was most seriously inaccurate for intensely selected populations which recombined freely. Intense selection generates more disequilibrium and free recombination allows more recovery in a generation of segregation.

LITERATURE CITED

Baker, L. H. and R. E. Comstock. 1961. Linkage and heterozygosity in finite populations. Mimeographed. Johnston, Iowa, Hy-Line Poultry Farms.

Barker, J. S. F. 1958. Simulation of genetic systems by automatic digital computers. III. Selection between alleles at an autosomal locus. Austrialian Journal of Biological Sciences 11: 603-612.

Berge, S. 1961. The historical development of animal breeding. In Schilling, E., ed. Schriftenreihe des Max-Panck-Instituts für Tierzucht und Tierernährung, Special Volume, 1961. pp. 109-127. Mariensee, Germany, Max-Planck-Gesellschaft-Dokumentationsstelle.

Bohidar, N. R. 1960. The role of sex-linked genes in quantitative inheritance. Unpublished Ph. D. thesis. Ames, Iowa, Library, Iowa State University of Science and Technology.

Bravais, A. 1846. Analyse mathématique sur les probabilités des erreurs de situation d'un point. Académic Royale des Sciences de L'Institute de France, Sciences Mathematiques et Physiques Memoires 9: 255-332.

Brownlee, J. 1910. The significance of the correlation coefficient when applied to Mendelian distribution. Royal Society of Edinburgh Proceedings 30: 473-507.

Cochran, W. G. 1951. Improvement by means of selection. Berkeley Symposium on Mathematical Statistics and Probability Proceedings 2: 449-470.

Cockerham, C. C. 1952. Genetic covariation among characteristics of swine. Unpublished Ph. D. thesis. Ames, Iowa, Library, Iowa State University of Science and Technology.

Cockerham, C. C. 1954. An extension of the concept of partitioning hereditary variance for analysis of covariances among relatives when epistasis is present. Genetics 39: 859-882.

Cockerham, C. C. 1956. Effects of linkage on the covariance between relatives. Genetics 41: 138-141.

Crow, J. F. 1954. Breeding structure of populations. 2. Effective population number. In Kempthorne, O., T. A. Bancroft, J. W. Gowen, and J. L. Lush, eds. Statistics and mathematics in biology. pp. 543-556. Ames, Iowa, Iowa State College Press.

Engeler, W. 1936. Die Entwicklung des Herdebuchwesens unter dem Einfluss der Lehren von der Vererbung and Züchtung bei den landwirtschaftlichen Haustieren. In Neue forschungen in tierzucht und abstammungslehre. pp. 39-70. Bern, Switzerland, Verbandsdruckerei A G.

Fisher, R. A. 1918. The correlation between relatives on the supposition of Mendelian inheritance. Royal Society of Edinburgh Transactions 52: 399-433.

Fraser, A. S. 1957a. Simulation of genetic systems by automatic digital computers. I. Introduction. Australian Journal of Biological Sciences 10: 484-491.

Fraser, A. S. 1957b. Simulation of genetic systems by automatic digital computers. II. Effects of linkage on rates of advance under selection. Australian Journal of Biological Sciences 10: 492-499.

Fraser, A. S. 1960. Simulation of genetic systems by automatic digital computers. V. Linkage, dominance and epistasis. In Kempthorne, O., ed. Biometrical genetics. pp. 70-83. New York, New York, Pergamon Press.

Fraser, A. S. 1962. Simulation of genetic systems. Journal of Theoretical Biology 2: 829-346.

Galton, F. 1889. Natural inheritance. London, England, Macmillan and Co.

Galton, F. 1897. The average contribution of each several ancestor to the total heritage of the offspring. Royal Society of London Proceedings 61: 401-413.

Gauss, K. F. 1806. Theoria motus corporum coelestium in sectionibus conicis solem ambientium (Theory of the motion of the heavenly bodies moving about the sun in conic sections). Translated by C. H. Davis. Boston, Massachusetts, Little, Brown and Company.

Gill, J. L. 1963. Selection in simulated genetic populations. Unpublished Ph. D. thesis. Ames, Iowa, Library, Iowa State University of Science and Technology.

Haldane, J. B. S. 1926. A mathematical theory of natural and artificial selection. IV. Cambridge Philosophical Society Proceedings 23: 607-615.

Jennings, H. S. 1916. The numerical results of diverse systems of breeding. Genetics 1: 53-89.

Jennings, H. S. 1917. The numerical results of diverse systems of breeding with respect to two pairs of characters linked or independent, with special relation to the effects of linkage. Genetics 2: 97-154.

Jones, R. M. 1960. Linkage distributions and epistacy in quantitative inheritance. Heredity 15: 153-159.

Kempthorne, O. 1952. Design and analysis of experiments. New York, New York, John Wiley and Sons, Inc.

Kempthorne, O. 1954. The correlation between relatives in a random mating population. Royal Society of London Proceedings, Series B, 143: 103-113.

Kempthorne, O. 1955. The theoretical values of correlations between relatives in random mating populations. Genetics 40: 153-167.

Lush, J. L. 1948. The genetics of populations. Mimeographed. Ames, Iowa, Department of Animal Science, Iowa State University of Science and Technology.

Lush, J. L. 1961. The meaning and estimation of heritability. In Schilling, E., ed. Schriftenreihe des Max-Planck-Instituts für Tierzucht and Tierernährung. Special Volume, 1961. pp. 147-170. Mariensee, Germany, Max-Planck-Gesellschaft-Dokumentationsstelle.

Malécot, G. 1948. Les Mathématiques de l'hérédité. Paris, France, Masson et Cie.

Martin, F. G., Jr. and C. C. Cockerham. 1960. High speed selection studies. In Kempthorne, O., ed. Biometrical genetics. pp. 35-45. New York, New York, Pergamon Press.

Pearson, K. 1898. Mathematical contributions to the theory of evolution. On the laws of ancestral heredity. Royal Society of London Proceedings 62: 386-412.

Pearson, K. 1903. The law of ancestral heredity. Biometrika 2: 211-236.

Pearson, K. 1904a. Mathematical contributions to the theory of evolution. XII. On a generalized theory of alternative inheritance, with special reference to Mendel's Laws. Royal Society of London Philosophical Transactions, Series A, 203: 53-86.

Pearson, K. 1904b. A Mendelians view of the law of ancestral inheritance. Biometrika 3: 109-112.

Qureshi, A. W. 1963. A Monte Carlo evaluation of the role of finite population size and linkage in response to continuous mass selection. Technical Report MC 6, Mimeographed. Ames, Iowa, Statistical Laboratory, Iowa State University of Science and Technology.

Reeve, E. C. R. 1953. Studies in quantitative inheritance. III. Heritability and genetic correlation in progeny tests using different systems. Journal of Genetics 51: 520-542.

Robbins, R. B. 1917. Applications of mathematics to breeding problems. Genetics 2: 489-504.

Robbins, R. B. 1918a. Applications of mathematics to breeding problems. II. Genetics 3: 73-92.

Robbins, R. B. 1918b. Applications of mathematics to breeding problems. III. Genetics 3: 375-379.

Robertson, A. 1960. A theory of limits in artificial selection. Royal Society of Edinburgh Proceedings, Series B, 153: 234-249.

Robinson, H. F. and R. E. Comstock. 1955. Analysis of the genetic variability in corn with reference to probable effects of selection. Cold Spring Harbor Symposia on Quantitative Biology 20: 127-136.

Robinson, H. F., R. E. Comstock and P. H. Harvey. 1949. Estimates of heritability and degree of dominance in corn. Agronomy Journal 41: 353-359.

Schnell, F. W. 1961. Some general formulations of linkage effects in inbreeding. Genetics 46: 947-958.

Schnell, F. W. 1963. The covariance between relatives in the presence of linkage. National Academy of Sciences - National Research Council Publication 982.

Shewhart, W. A. 1926. Correction of data for errors of measurement. Bell System Technical Journal 5: 11-26.

Snow, E. C. 1910. On the determination of the chief correlations between collaterals in the case of a simple Mendelian population mating at random. Royal Society of London Proceedings, Series A, 83: 37-55.

Van Aarde, I. M. R. 1963. Covariances of relatives in random mating populations with linkage. Unpublished Ph. D. thesis. Ames, Iowa, Library, Iowa State University of Science and Technology.

Weckherlin, A. von. 1851. Landwirtschaftliche Tierproduktionslehre. Vol. 3. Schafzucht. second ed. Stuttgart. Original not available; cited in Engeler, W. 1936. Die Entwicklung des Herdebuchwesens unter dem Einfluss der Lehren von der Vererbung and Züchtung bei den landwirtschaftlichen Haustieren. In Neue forschungen in tierzucht und abstammungslehre. pp. 43. Bern, Switzerland, Verbandsdruckerei AG.

Weinberg, W. 1908a. Über Vererbungsgesetze beim Menschen. Zeitschift für induktive Abstammungs and Vererbungslehre 1: 377-392.

Weinberg, W. 1908b. Über Vererbungsgesetze beim Menschen. Zeitschrift für induktive Abstammungs und Vererbungslehre 1: 440-460.

Weinberg, W. 1909. Über Vererbungsgesetze beim Menschen. Zeitschrift für induktive Abstammungs und Vererbungslehre 2: 276-330.

Winsor, C. P. 1946. Which regression? Biometrics 2: 101-109.

Wright, S. 1921a. Systems of mating. I. The biometric relations between parent and offspring. Genetics 6: 111-123.

Wright, S. 1921b. Correlation and causation. Journal of Agricultural Research 10: 557-585.

Wright, S. 1934. The method of path coefficients. Annals of Mathematical Statistics 5: 161-215.

Yule, G. U. 1902. Mendel's Laws and their probable relations to intra-racial heredity. New Phytologist 1: 193-207, 222-238.

Yule, G. U. 1907. On the theory of inheritance of quantitative compound characters on the basis of Mendel's Laws: a preliminary note. International Conference on Genetics, London, 1906. Royal Horticultural Society 3: 140-142.

ACKNOWLEDGEMENTS

The author wishes to express his sincere appreciation to Dr. J. L. Lush for his guidance and training up to the final preparation of this manuscript, to Dr. L. N. Hazel for his guidance in developing the study and his attention to the final preparation of the manuscript, and to Professor Oscar Kempthorne and Dr. D. L. Harris for their critical review of the initial problem. The computation was supported by grant number NSF-G19218 from the National Science Foundation. The Regional Swine Breeding Laboratory of the United States Department of Agriculture, under the direction of Dr. C. E. Shelby, allowed the author time to pursue part of this study while employed as Geneticist.

APPENDIX A: THE CORRELATION BETWEEN FULL SIBS AND PATERNAL HALF SIBS WHEN PARENTS ARE NOT CHOSEN AT RANDOM

Reeve (1953) used the method of path coefficients to develop expressions of the correlations between relatives for a number of different mating systems. He included the selection of parents and phenotypic assortative mating by taking advantage of the results of Wright (1921b, 1934) for the case of random mating of parents chosen at random. In considering the analysis of variance and covariance of progeny families, Reeve considered only the variance and covariances between and with full-sib families. Some further refinements are developed here for full-sib and paternal half-sib families (Design I, Robinson et al., 1949).

The definitions and symbols used by Reeve will be used here, despite some inconvenience which results. He was concerned with selection and phenotypic assortative mating for the express purpose of increasing the variability among parents. He therefore defined the factor $(1 + K)$ as representing the increase in the phenotypic variance among selected parents. Such definition is confusing when selection has reduced the phenotypic variance among parents and one must remember that K is negative. We will use $(1 + K)$ for selected male parents, use $(1 + K')$ for selected female parents, and define $(1 + \bar{K})$ to be the arithmetic mean of $(1 + K)$ and $(1 + K')$. The subscript o indicates the parameters of the selected population of parents, the subscript 1 indicates the parameters of their progeny, and parameters without subscript refer to the unselected population. The superscript (')

will be used to distinguish female from male. For the sake of clarity, for the purposes intended here, it will be assumed that there is no assortative mating and no correlation between uniting gametes (no inbreeding).

The primary effect of selection, by definition, is that

$$\sigma^2\{P\} = (1 + K)\sigma^2\{P\} \text{ and } \sigma^2\{P'_o\} = (1 + K')\sigma^2\{P\}.$$

It is well-known that if $\underline{x}$ and $\underline{y}$ are two linearly correlated variables, selection on $\underline{x}$ has the following results on the variance of $\underline{y}$.

$$\sigma^2\{y_o\} = \left[1 - r^2 + r^2 \frac{\sigma^2\{x_o\}}{\sigma^2\{x\}}\right] \sigma^2\{y\}$$

and

$$r_o = \frac{\sigma\{x_o\}\sigma\{y\}}{\sigma\{x\}\sigma\{y_o\}} r$$

The subscript $\underline{o}$ indicates the selected population and r and r_o are the linear correlation coefficients in the unselected and selected populations.

It is assumed that all effects are additive and that the effects of heritability and environment are uncorrelated ($P = H + E$). In Figures 1 and 2, $\underline{e}$ and $\underline{h}$ are the path coefficients from $\underline{E}$ to $\underline{P}$ and from $\underline{H}$ to $\underline{P}$, so that $\underline{e}^2$ and $\underline{h}^2$ measure the fractions of the phenotypic variance resulting from environmental and additively genetic causes. $\underline{G}$ and

G' represent the male and female germ cells. The correlation (r_{PH}) between phenotype (P) and the genetic effect (H) is h, the correlation between phenotype (P) and the environmental effect (E) is e. Assuming that uniting gametes are uncorrelated, $\sigma^2\{G\} = 1/2\sigma^2\{H\}$ and the correlation between phenotype (P) and the effect of a gamete (G) is $h/\sqrt{2}$. We can then show that:

$$\sigma^2\{H_o\} = (1 + Kh^2)\sigma^2\{H\},$$

$$\sigma^2\{H'_o\} = (1 + K'h^2)\sigma^2\{H\},$$

$$\sigma^2\{E_o\} = (1 + Ke^2)\sigma^2\{E\},$$

$$\sigma^2\{E'_o\} = (1 + K'e^2)\sigma^2\{E\},$$

$$\sigma^2\{G_o\} = (1 + Kh^2/2)\sigma^2\{H\}$$

$$\sigma^2\{G'_o\} = (1 + K'h^2/2)\sigma^2\{H\},$$

$$\sigma^2\{H\} = \sigma^2\{G_o\} + \sigma^2\{G'_o\} = (1 + \bar{K}h^2/2)\sigma^2\{H\},$$

$$\sigma^2\{P_1\} = \sigma^2\{H_1\} + \sigma^2\{E_1\} = (1 + \bar{K}h^4/2)\sigma^2\{P\} \text{ if } \sigma^2\{E_1\} = \sigma^2\{E\}.$$

From Figure 1 we see that the correlation between paternal half-sibs is;

$$r_{PHS} = \frac{h^2}{4}\left[\frac{1 + Kh^2}{1 + \bar{K}h^4/2}\right]$$

and from Figure 2 the correlation between full-sibs is

$$r_{FS} = \frac{h^2}{2}\left[\frac{1 + \bar{K}h^2}{1 + \bar{K}h^4/2}\right].$$

The analyses of full-sib families and paternal half-sib families (Design I, Robinson et al., 1949) gives estimates of the sire component of variance (S), the dam component of variance (D), and the within full-sib family component of variance (W). The sire and dam components are measures of the progeny variance arising from genetic differences among male parents and among female parents mated to the same male. When parents have not been chosen at random, the value of S would be expected to be $E(S) = 1/4(1 + Kh^2)\sigma^2\{H\}$ and the value of D would be expected to be $E(D) = 1/4(1 + K'h^2)\sigma^2\{H\}$. The expected value of the sum of the three components of variance, S + D + W, is

$$\sigma^2\{P_1\} = (1 + \bar{K}h/2)\sigma^2\{P\}.$$

We then find the expected value of W to be

$$E(W) = 1/2\sigma^2\{H\} + \sigma^2\{E_1\}$$

or $1/2\sigma^2\{H\}$ if there is no environmental variance.

APPENDIX B: TABLES

Table 1. Average recombination fractions ($\bar{r}$) for linkage parameters of some previous Monte Carlo studies and the present study

r	$\bar{r}$	Number of autosomes	Loci per autosome	Reference
0.500	0.500	2	3	Fraser (1957b)
0.250	0.417			
0.050	0.326			
0.025	0.313			
0.005	0.303			
0.500	0.500	1	5	Martin and Cockerham (1960)
0.010	0.020			
0.500	0.500	1	20	
0.300	0.468			
0.100	0.341			
0.010	0.064			
0.500	0.500	1	35	Baker and Comstock (1961)
0.010	0.102			
0.500	0.500	8	5	Gill (1963)
0.200	0.479			
0.050	0.458			
0.005	0.450			
0.500	0.500	4	10	Qureshi (1963)
0.050	0.420			
0.005	0.389			
0.500	0.500	1	40	
0.010	0.114			

Table 2. Means of the ratios of predicted to achieved genetic improvement for "Regression methods"--no environmental variance

Gen.	000	001	010	011	100	101	110	111
	DOS							
1	1.016	1.020	0.974	1.000	1.012	0.988	1.001	0.994
2	1.011	1.040	1.039	1.010	0.994	1.009	1.003	0.986
3	0.994	0.977	1.010	1.021	1.005	1.020	1.000	1.000
4	0.966	0.992	0.993	0.993	1.000	0.994	1.015	1.006
5	1.000	1.001	1.012	1.017	0.995	0.993	0.995	0.986
Ave.	0.997	1.006	1.005	1.008	1.001	1.001	1.003	0.994
	DO							
1	1.003	1.018	0.981	1.002	1.002	0.996	0.999	0.985
2	1.012	1.045	1.008	1.013	1.009	1.010	1.009	1.005
3	0.993	0.988	0.996	1.005	1.004	1.017	0.987	0.994
4	0.959	0.973	1.007	0.979	0.999	0.994	1.016	1.000
5	0.997	1.003	1.016	1.004	0.994	0.988	0.990	0.988
Ave.	0.993	1.005	1.001	1.001	1.002	1.001	1.000	0.994
	MPO							
1	1.008	1.007	0.985	0.997	1.001	0.992	0.998	0.992
2	1.004	1.026	1.013	1.017	1.003	1.011	0.997	0.989
3	0.997	0.974	0.975	1.005	1.003	1.015	0.994	0.992
4	0.982	0.982	1.012	0.991	0.999	0.998	1.011	0.996
5	1.005	1.017	1.012	1.028	0.999	1.000	1.000	0.985
Ave.	0.999	1.001	0.999	1.008	1.001	1.003	1.000	0.991

Table 3. Main effect means of the ratios of predicted to achieved genetic improvement for "Regression methods"--no environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	DOS						
1	1.003	0.999	1.009	0.992	1.001	1.000	1.001
2	1.025	0.998	1.014	1.009	1.012	1.011	1.011
3	1.001	1.006	1.999	1.008	1.002	1.004	1.003
4	0.986	1.004	0.988	1.002	0.993	0.996	0.995
5	1.007	0.992	0.997	1.002	1.000	0.999	1.000
Ave.	1.004	1.000	1.001	1.003	1.002	1.002	1.002
	DO						
1	1.001	0.996	1.005	0.991	0.996	1.000	0.998
2	1.019	1.008	1.019	1.009	1.009	1.018	1.014
3	0.996	1.001	1.001	0.996	0.995	1.001	0.998
4	0.980	1.002	0.981	1.000	0.995	0.987	0.991
5	1.005	0.990	0.995	0.999	0.999	0.996	0.997
Ave.	1.000	0.999	1.000	0.999	0.999	1.000	1.000
	MPO						
1	0.999	0.996	1.002	0.993	0.998	0.997	0.998
2	1.015	1.000	1.011	1.004	1.004	1.011	1.007
3	0.988	1.001	0.997	0.991	0.992	0.996	0.994
4	0.992	1.001	0.990	1.002	1.001	0.992	0.996
5	1.016	0.996	1.005	1.006	1.004	1.008	1.006
Ave.	1.002	0.999	1.001	0.999	1.000	1.001	1.000

Table 4. Selection x linkage means of the ratios of predicted to achieved genetic improvement for "Regression methods--no environmental variance

Gen.	0.0	0.1	1.0	1.0	...
	DOS				
1	0.995	1.010	1.006	0.991	1.001
2	1.025	1.025	0.998	0.998	1.011
3	1.002	0.999	1.002	1.010	1.003
4	0.979	0.993	1.008	1.000	0.995
5	1.006	1.009	0.995	0.990	1.000
Ave.	1.001	1.007	1.002	0.997	1.002
	DO				
1	0.992	1.010	1.000	0.991	0.998
2	1.010	1.029	1.009	1.007	1.014
3	0.995	0.996	0.996	1.005	0.998
4	0.983	0.976	1.007	0.997	0.991
5	1.007	1.004	0.992	0.988	0.997
Ave.	0.997	1.003	1.001	0.998	1.000
	MPO				
1	0.996	1.002	1.000	0.992	0.998
2	1.008	1.022	1.000	1.000	1.007
3	0.986	0.990	0.998	1.003	0.994
4	0.997	0.986	1.005	0.997	0.996
5	1.008	1.023	0.999	0.993	1.006
Ave.	0.999	1.004	1.000	0.997	1.000

Table 5. Means of the ratios of predicted to achieved genetic improvement for "Regression methods"--environmental variance

Gen.	000	001	010	011	100	101	110	111
	DOS							
1	1.037	0.955	1.048	1.132	0.984	0.981	1.022	0.981
2	1.117	1.053	0.934	1.067	1.026	1.050	1.033	1.040
3	0.995	1.143	1.023	1.167	1.002	1.019	1.045	0.970
4	0.863	1.006	0.950	0.940	1.035	1.046	1.065	0.927
5	0.842	1.092	0.883	0.912	1.064	0.991	1.115	1.049
Ave.	0.971	1.050	0.968	1.044	1.022	1.017	1.056	0.993
	DO							
1	1.036	1.015	1.114	1.114	0.961	0.998	1.034	0.960
2	1.140	1.058	0.998	1.047	1.004	1.035	0.975	1.063
3	0.971	1.129	0.967	1.182	1.002	1.004	1.069	0.977
4	0.780	0.928	0.880	0.998	1.040	1.010	0.993	0.916
5	0.815	1.047	0.903	0.976	1.059	0.983	1.126	1.080
Ave.	0.948	1.035	0.973	1.063	1.013	1.006	1.039	0.999
	MPO							
1	1.105	1.056	1.150	1.186	0.943	1.008	1.034	0.951
2	1.112	0.952	1.023	1.043	1.033	1.049	1.006	1.053
3	0.982	1.114	0.997	1.165	0.993	1.026	1.075	0.989
4	0.945	0.997	0.989	1.062	1.009	1.025	0.974	0.898
5	0.909	1.042	0.797	0.916	1.053	1.010	1.104	1.093
Ave.	1.010	1.032	0.991	1.074	1.006	1.024	1.038	0.997

Table 6. Main effect means of the ratios of predicted to achieved genetic improvement for "Regression methods"--environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	DOS						
1	1.043	0.992	0.989	1.046	1.023	1.012	1.017
2	1.043	1.037	1.061	1.019	1.028	1.053	1.040
3	1.082	1.009	1.040	1.051	1.016	1.075	1.045
4	0.940	1.018	0.987	0.971	0.978	0.980	0.979
5	0.932	1.055	0.997	0.990	0.976	1.011	0.993
Ave.	1.008	1.022	1.015	1.015	1.004	1.026	1.015
	DO						
1	1.070	0.988	1.002	1.056	1.036	1.022	1.029
2	1.061	1.019	1.059	1.021	1.029	1.051	1.040
3	1.062	1.013	1.026	1.049	1.002	1.073	1.038
4	0.897	0.990	0.940	0.947	0.923	0.963	0.943
5	0.935	1.062	0.976	1.021	0.976	1.022	0.999
Ave.	1.005	1.014	1.001	1.019	0.993	1.026	1.010
	MPO						
1	1.124	0.984	1.028	1.080	1.058	1.050	1.054
2	1.032	1.035	1.037	1.031	1.043	1.024	1.034
3	1.064	1.021	1.029	1.056	1.012	1.074	1.043
4	0.998	0.977	0.994	0.981	0.979	0.995	0.987
5	0.916	1.065	1.003	0.978	0.966	1.015	0.991
Ave.	1.027	1.016	1.018	1.025	1.012	1.032	1.022

Table 7. Selection x linkage means of the ratios of predicted to achieved genetic improvement for "Regression methods"--environmental variance

Gen.	0.0	0.1	1.0	1.1	...
	DOS				
1	1.043	1.043	1.003	0.981	1.017
2	1.026	1.060	1.030	1.045	1.040
3	1.009	1.155	1.024	0.994	1.045
4	0.906	0.973	1.050	0.987	0.979
5	0.862	1.002	1.090	1.020	0.993
Ave.	0.969	1.047	1.039	1.005	1.015
	DO				
1	1.075	1.065	0.998	0.979	1.029
2	1.069	1.052	0.989	1.049	1.040
3	0.969	1.155	1.035	0.991	1.038
4	0.830	0.963	1.016	0.963	0.943
5	0.859	1.011	1.093	1.032	0.999
Ave.	0.961	1.049	1.026	1.003	1.010
	MPO				
1	1.128	1.121	0.988	0.980	1.054
2	1.067	0.997	1.020	1.051	1.034
3	0.989	1.140	1.034	1.008	1.043
4	0.967	1.029	0.991	0.962	0.987
5	0.853	0.979	1.078	1.051	0.991
Ave.	1.001	1.053	1.022	1.010	1.022

Table 8. Means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--no environmental variance

Gen.	000	001	010	011	100	101	110	111
	PHS							
1	0.206	0.216	0.245	0.298	0.183	0.234	0.252	0.210
2	0.251	0.196	0.179	0.181	0.217	0.196	0.215	0.262
3	0.186	0.180	0.204	0.174	0.165	0.233	0.136	0.161
4	0.215	0.180	0.250	0.170	0.147	0.202	0.137	0.171
5	0.231	0.195	0.218	0.193	0.131	0.201	0.106	0.176
Ave.	0.218	0.194	0.219	0.203	0.168	0.213	0.169	0.196
	PHSA							
1	1.018	0.997	0.968	0.963	1.053	0.953	0.973	1.019
2	1.060	1.111	1.056	1.086	1.003	1.025	1.042	0.976
3	1.074	1.064	1.035	1.104	1.008	1.030	1.042	1.078
4	0.998	1.099	1.063	1.080	1.027	1.028	0.979	1.036
5	1.049	1.054	1.053	1.061	1.041	0.972	1.002	1.052
Ave.	1.040	1.065	1.035	1.059	1.027	1.002	1.008	1.032
	FS							
1	0.327	0.317	0.320	0.337	0.718	0.751	0.752	0.712
2	0.309	0.261	0.287	0.245	0.568	0.653	0.571	0.640
3	0.275	0.225	0.289	0.239	0.632	0.640	0.633	0.613
4	0.294	0.239	0.295	0.239	0.611	0.606	0.638	0.620
5	0.302	0.254	0.282	0.252	0.633	0.607	0.673	0.607
Ave.	0.301	0.259	0.294	0.262	0.633	0.651	0.653	0.638
	FSA							
1	0.982	0.976	0.973	0.983	1.002	1.010	1.006	0.983
2	1.082	1.134	1.074	1.133	0.732	0.865	0.717	0.838
3	1.101	1.127	1.013	1.145	0.846	0.827	0.847	0.786
4	1.035	1.140	1.106	1.120	0.813	0.791	0.874	0.810
5	1.077	1.100	1.111	1.114	0.840	0.789	0.918	0.787
Ave.	1.055	1.095	1.056	1.099	0.847	0.856	0.872	0.841

Table 9. Main effect means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--no environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	PHS						
1	0.241	0.219	0.210	0.251	0.222	0.239	0.230
2	0.202	0.222	0.215	0.209	0.216	0.209	0.212
3	0.186	0.174	0.191	0.169	0.172	0.187	0.180
4	0.204	0.164	0.186	0.182	0.187	0.181	0.184
5	0.209	0.154	0.190	0.173	0.172	0.191	0.182
Ave.	0.208	0.187	0.198	0.197	0.194	0.201	0.198
	PHSA						
1	0.986	1.000	1.005	0.981	1.003	0.983	0.993
2	1.078	1.011	1.049	1.040	1.040	1.049	1.045
3	1.069	1.040	1.044	1.065	1.040	1.069	1.054
4	1.060	1.017	1.038	1.039	1.017	1.061	1.039
5	1.054	1.017	1.029	1.042	1.036	1.035	1.036
Ave.	1.050	1.017	1.033	1.033	1.027	1.039	1.033
	FS						
1	0.325	0.733	0.528	0.530	0.529	0.529	0.529
2	0.275	0.608	0.448	0.436	0.433	0.450	0.442
3	0.257	0.630	0.443	0.443	0.457	0.429	0.443
4	0.267	0.619	0.438	0.448	0.460	0.426	0.443
5	0.273	0.630	0.449	0.454	0.473	0.430	0.451
Ave.	0.279	0.644	0.461	0.462	0.470	0.453	0.462
	FSA						
1	0.978	1.000	0.992	0.986	0.991	0.988	0.989
2	1.106	0.788	0.953	0.940	0.901	0.992	0.947
3	1.097	0.827	0.976	0.948	0.952	0.971	0.962
4	1.100	0.822	0.945	0.977	0.957	0.965	0.961
5	1.101	0.834	0.951	0.983	0.986	0.948	0.967
Ave.	1.076	0.854	0.963	0.967	0.957	0.973	0.965

Table 10. Selection x linkage means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--no environmental variance

Gen.	0.0	0.1	1.0	1.1	...
	PHS				
1	0.226	0.257	0.217	0.222	0.230
2	0.215	0.189	0.216	0.229	0.212
3	0.195	0.177	0.150	0.197	0.180
4	0.233	0.175	0.142	0.187	0.184
5	0.225	0.194	0.119	0.189	0.182
Ave.	0.219	0.198	0.169	0.205	0.198
	PHSA				
1	0.993	0.980	1.013	0.986	0.993
2	1.058	1.099	1.022	1.000	1.045
3	1.055	1.084	1.025	1.054	1.054
4	1.030	1.090	1.003	1.032	1.039
5	1.051	1.058	1.021	1.012	1.036
Ave.	1.037	1.062	1.017	1.017	1.033
	FS				
1	0.323	0.327	0.735	0.732	0.529
2	0.298	0.253	0.569	0.647	0.442
3	0.282	0.232	0.633	0.627	0.443
4	0.294	0.239	0.625	0.613	0.443
5	0.292	0.253	0.653	0.607	0.451
Ave.	0.298	0.261	0.643	0.645	0.462
	FSA				
1	0.978	0.979	1.004	0.997	0.989
2	1.078	1.133	0.724	0.851	0.947
3	1.057	1.136	0.847	0.806	0.962
4	1.071	1.130	0.844	0.800	0.961
5	1.094	1.107	0.879	0.788	0.967
Ave.	1.055	1.097	0.860	0.849	0.965

Table 11. Means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--environmental variance

Gen.	000	001	010	011	100	101	110	111
	PHS							
1	0.649	0.617	0.654	0.613	0.482	0.581	0.666	0.538
2	0.585	0.598	0.582	0.530	0.583	0.665	0.478	0.585
3	0.577	0.708	0.652	0.545	0.537	0.710	0.552	0.661
4	0.681	0.691	0.623	0.671	0.505	0.629	0.499	0.593
5	0.587	0.638	0.590	0.584	0.594	0.619	0.585	0.634
Ave.	0.616	0.650	0.620	0.589	0.540	0.641	0.556	0.602
	PHSA							
1	1.079	0.985	1.081	0.997	0.730	0.870	1.015	0.829
2	0.894	0.870	0.894	0.717	0.882	1.122	0.667	0.856
3	0.819	1.131	0.970	0.755	0.784	1.115	0.805	1.099
4	0.995	1.085	0.847	1.036	0.721	0.942	0.675	0.880
5	0.745	0.884	0.716	0.773	0.901	0.942	0.774	0.904
Ave.	0.906	0.991	0.902	0.856	0.804	0.998	0.787	0.914
	FS							
1	0.671	0.660	0.676	0.654	0.770	0.840	0.847	0.808
2	0.698	0.635	0.645	0.637	0.787	0.884	0.718	0.834
3	0.663	0.675	0.697	0.625	0.783	0.855	0.795	0.795
4	0.711	0.663	0.668	0.662	0.776	0.821	0.817	0.773
5	0.667	0.661	0.726	0.679	0.844	0.824	0.903	0.829
Ave.	0.684	0.659	0.682	0.651	0.792	0.845	0.816	0.808
	FSA							
1	1.079	1.052	1.087	1.003	0.915	0.995	1.003	0.953
2	1.040	0.897	0.940	0.873	0.913	1.042	0.832	0.983
3	0.923	0.939	0.941	0.825	0.920	1.014	0.932	0.927
4	0.956	0.905	0.836	0.889	0.909	0.978	0.964	0.909
5	0.864	0.873	0.897	0.895	0.975	0.963	1.046	0.974
Ave.	0.972	0.933	0.940	0.897	0.926	0.998	0.955	0.949

Table 12. Main effect means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	PHS						
1	0.634	0.567	0.582	0.618	0.613	0.587	0.600
2	0.574	0.578	0.608	0.544	0.557	0.595	0.576
3	0.621	0.615	0.633	0.603	0.580	0.656	0.618
4	0.667	0.556	0.627	0.597	0.577	0.646	0.612
5	0.600	0.608	0.609	0.598	0.589	0.619	0.604
Ave.	0.619	0.585	0.612	0.592	0.583	0.621	0.602
	PHSA						
1	1.035	0.861	0.916	0.980	0.976	0.920	0.948
2	0.844	0.882	0.942	0.784	0.834	0.891	0.863
3	0.919	0.951	0.962	0.907	0.844	1.025	0.935
4	0.991	0.804	0.936	0.860	0.810	0.986	0.898
5	0.779	0.880	0.868	0.792	0.784	0.876	0.830
Ave.	0.914	0.876	0.925	0.865	0.850	0.940	0.895
	FS						
1	0.665	0.816	0.735	0.746	0.741	0.740	0.741
2	0.654	0.806	0.751	0.708	0.712	0.747	0.730
3	0.665	0.807	0.744	0.728	0.735	0.738	0.736
4	0.676	0.797	0.743	0.730	0.743	0.730	0.736
5	0.686	0.850	0.751	0.784	0.788	0.748	0.768
Ave.	0.669	0.815	0.749	0.739	0.744	0.741	0.742
	FSA						
1	1.055	0.966	1.010	1.012	1.021	1.001	1.011
2	0.937	0.943	0.973	0.907	0.931	0.949	0.940
3	0.907	0.948	0.949	0.906	0.929	0.926	0.928
4	0.896	0.940	0.937	0.899	0.916	0.920	0.918
5	0.882	0.989	0.919	0.953	0.945	0.926	0.936
Ave.	0.936	0.957	0.958	0.935	0.949	0.944	0.947

Table 13. Selection x linkage means of the ratios of predicted to achieved genetic improvement for "Variance component methods"--environmental variance

Gen.	0.0	0.1	1.0	1.1	...
	PHS				
1	0.652	0.615	0.574	0.559	0.600
2	0.584	0.564	0.531	0.625	0.576
3	0.614	0.627	0.545	0.686	0.618
4	0.652	0.681	0.502	0.611	0.612
5	0.589	0.611	0.590	0.626	0.604
Ave.	0.618	0.620	0.548	0.622	0.602
	PHSA				
1	1.080	0.991	0.872	0.849	0.948
2	0.894	0.793	0.775	0.989	0.863
3	0.895	0.943	0.794	1.107	0.935
4	0.921	1.061	0.698	0.911	0.898
5	0.730	0.829	0.838	0.923	0.830
Ave.	0.904	0.923	0.795	0.956	0.895
	FS				
1	0.673	0.657	0.808	0.824	0.741
2	0.671	0.636	0.753	0.859	0.730
3	0.680	0.650	0.789	0.825	0.736
4	0.689	0.662	0.797	0.797	0.736
5	0.701	0.670	0.874	0.826	0.768
Ave.	0.683	0.655	0.804	0.826	0.742
	FSA				
1	1.083	1.027	0.959	0.974	1.011
2	0.990	0.885	0.873	1.012	0.940
3	0.932	0.882	0.926	0.970	0.928
4	0.896	0.897	0.936	0.944	0.918
5	0.880	0.884	1.010	0.968	0.936
Ave.	0.956	0.915	0.941	0.974	0.947

Table 14. Analyses of variance of the ratios of predicted to achieved genetic improvement for "Regression methods"--no environmental variance

SOV	d.f.	MPO	DO	DOS
Generation	4	.00446	.00916	.00475
Selection	1	.00148	.00010	.00332
Population	1	.00049	.00021	.00028
Linkage	1	.00012	.00025	.00005
SXP	1	.00376	.00136	.00226
SXL	1	.00296	.00333	.00428
PXL	1	.00026	.00331	.00200
SXPXL	1	.00323	.00070	.00004
GXS	4	.00664[a]	.00734	.00961
GXP	4	.00233	.00552	.00472
GXL	4	.00131	.00165	.00008
GXSXP	4	.00162	.00289	.00542
GXSXL	4	.00080	.00170	.00210
GXPXL	4	.00206	.00120	.00472
GXSXPXL	4	.00173	.00108	.00160
ERROR	600	.00258	.00520	.00494

[a] $P < .05$

Table 15. Analyses of variance of the ratios of predicted to achieved genetic improvement for "Regression methods"--environmental variance

SOV	d.f.	MPO	DO	DOS
Generation	4	.12117	.21178[a]	.10635
Selection	1	.01862	.01442	.03281
Population	1	.00790	.05121	.00000
Linkage	1	.06482	.17025	.07659
SXP	1	.00332	.01084	.00379
SXL	1	.16636	.50527[b]	.49674[b]
PXL	1	.00004	.00861	.03697
SXPXL	1	.14622	.01318	.02972
GXS	4	.34941[c]	.28085[b]	.22462[b]
GXP	4	.03297	.04254	.04370
GXL	4	.03968	.03198	.02384
GXSXP	4	.11557	.07782	.06214
GXSXL	4	.10527	.15244	.06096
GXPXL	4	.03039	.03161	.08793
GXSXPXL	4	.02127	.03791	.06652
ERROR	600	.05593	.06902	.06525

[a] $P < .05$

[b] $P < .01$

[c] $P < .001$

Table 16. Analyses of variance of the ratios of predicted to achieved genetic improvement for "Variance component methods"--no environmental variance

SOV	d.f.	FS	PHS	d.f.	FSA	PHSA
Generation	4	.18419[c]	.06567[c]	3	.00965	.00892
Selection	1	21.27467[c]	.07591[b]	1	10.28081[c]	.24885[c]
Population	1	.00018	.00032	1	.00463	.00542
Linkage	1	.04887[c]	.00977	1	.05135[a]	.05298
SXP	1	.00131	.00773	1	.00107	.00063
SXL	1	.06095[c]	.12458[c]	1	.12870[c]	.02353
PXL	1	.00574	.00096	1	.01489	.00282
SXPXL	1	.01913[a]	.00697	1	.01595	.00806
GXS	4	.02537[c]	.02707[a]	3	.01764	.00807
GXP	4	.00218	.02009	3	.03002[a]	.00536
GXL	4	.01965[c]	.00559	3	.09221[c]	.01306
GXSXP	4	.00429	.02129	3	.00198	.00983
GXSXL	4	.02229[c]	.02270	3	.06575[c]	.00579
GXPXL	4	.00040	.01328	3	.01597	.02283
GXSXPXL	4	.00245	.00813	3	.01335	.02535
ERROR	600	.00361	.00978	480	.00836	.01424

[a] $P < .05$

[b] $P < .01$

[c] $P < .001$

Table 17. Analyses of variance of the ratios of predicted to achieved genetic improvement for "Variance component methods"--environmental variance

SOV	d.f.	FS	PHS	d.f.	FSA	PHSA
Generation	4	.02831	.03304	3	.01188	.26075
Selection	1	3.42227[c]	.18483[a]	1	.31200[b]	.00212
Population	1	.00482	.06290	1	.10106	1.07110[a]
Linkage	1	.00135	.22461[a]	1	.00032	2.04541[c]
SXP	1	.00015	.01154	1	.01146	.00052
SXL	1	.10171[a]	.20720[a]	1	.17807[a]	.81935[a]
PXL	1	.04445	.14575	1	.02474	.22819
SXPXL	1	.03005	.00135	1	.04420	.21195
GXS	4	.00835	.08761	3	.05746	.50506
GXP	4	.02620	.04216	3	.05997	.06705
GXL	4	.02372	.05231	3	.00735	.12194
GXSXP	4	.00447	.01719	3	.00720	.11244
GXSXL	4	.02772	.02166	3	.12899[a]	.19274
GXPXL	4	.01577	.03864	3	.03724	.16649
GXSXPXL	4	.00670	.04829	3	.02364	.12532
ERROR	600	.01804	.04061	480	.04649	.17470

[a] $P < .05$

[b] $P < .01$

[c] $P < .001$

Table 18. Means of the variability among selected parents expressed as fractions of the variance in the unselected population with no environmental variance

Gen.	000	001	010	011	100	101	110	111
	$1 + K$							
1	.135	.131	.139	.166	.184	.183	.196	.186
2	.188	.179	.152	.163	.177	.179	.207	.204
3	.154	.152	.144	.167	.146	.204	.155	.187
4	.141	.166	.180	.154	.144	.190	.115	.176
5	.169	.155	.169	.152	.137	.168	.111	.179
Ave.	.157	.157	.157	.160	.158	.185	.157	.186
	$1 + \overline{K}$							
1	.192	.186	.190	.202	.557	.598	.602	.567
2	.228	.219	.208	.212	.572	.580	.602	.591
3	.211	.202	.190	.216	.568	.595	.568	.599
4	.195	.212	.224	.204	.567	.577	.546	.579
5	.214	.200	.223	.204	.572	.583	.566	.580
Ave.	.208	.204	.207	.208	.567	.587	.577	.583

Table 19. Main effect means of the variability among selected parents expressed as fractions of the variance in the unselected population with no environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	$1 + K$						
1	.143	.187	.158	.172	.164	.166	.165
2	.171	.192	.181	.182	.181	.181	.181
3	.154	.173	.164	.163	.150	.177	.164
4	.160	.156	.160	.156	.145	.171	.158
5	.161	.148	.157	.153	.146	.163	.155
Ave.	.158	.171	.164	.165	.157	.171	.165
	$1 + \overline{K}$						
1	.192	.581	.383	.390	.385	.388	.387
2	.217	.586	.400	.403	.402	.400	.401
3	.205	.582	.394	.393	.384	.403	.393
4	.209	.567	.388	.388	.383	.393	.388
5	.210	.575	.392	.393	.394	.392	.392
Ave.	.206	.578	.391	.394	.390	.395	.392

Table 20. Selection x linkage means of the variability among selected parents expressed as fractions of the variance in the unselected population with no environmental variance

Gen.	0.0	0.1	1.0	1.1	...
	$1 + K$				
1	.137	.148	.190	.184	.165
2	.170	.171	.192	.192	.181
3	.149	.160	.150	.195	.164
4	.160	.160	.130	.183	.158
5	.169	.154	.124	.173	.155
Ave.	.157	.159	.157	.185	.165
	$1 + \overline{K}$				
1	.191	.194	.580	.582	.387
2	.218	.215	.587	.585	.401
3	.200	.209	.568	.597	.393
4	.210	.208	.557	.578	.388
5	.218	.202	.569	.582	.392
Ave.	.207	.206	.572	.585	.392

Table 21. Means of the variability among selected parents expressed as fractions of the variance in the unselected population with environmental variance

Gen.	000	001	010	011	100	101	110	111
	$1 + K$							
1	.140	.135	.152	.149	.162	.180	.188	.186
2	.139	.142	.139	.138	.183	.175	.170	.173
3	.134	.141	.116	.143	.155	.184	.172	.174
4	.130	.130	.150	.133	.182	.155	.170	.180
5	.131	.120	.124	.147	.174	.176	.171	.158
Ave.	.135	.134	.136	.142	.171	.174	.174	.174
	$1 + \overline{K}$							
1	.187	.179	.197	.194	.588	.604	.605	.617
2	.187	.189	.185	.185	.593	.602	.579	.595
3	.186	.181	.176	.192	.581	.580	.575	.587
4	.177	.183	.187	.184	.577	.572	.570	.570
5	.178	.179	.180	.190	.602	.579	.590	.563
Ave.	.183	.182	.184	.189	.588	.587	.584	.586

Table 22. Main effect means of the variability among selected parents expressed as fractions of the variance in the unselected population with environmental variance

Gen.	0..	1..	.0.	.1.	..0	..1	...
	$1 + K$						
1	.144	.179	.154	.169	.160	.162	.161
2	.140	.175	.160	.155	.158	.157	.157
3	.134	.171	.153	.151	.144	.160	.152
4	.135	.172	.149	.158	.158	.149	.154
5	.130	.170	.150	.150	.150	.150	.150
Ave.	.137	.173	.153	.157	.154	.156	.155
	$1 + \overline{K}$						
1	.189	.603	.389	.403	.394	.398	.396
2	.186	.592	.393	.386	.386	.393	.389
3	.184	.581	.382	.382	.380	.385	.382
4	.182	.572	.377	.377	.378	.377	.377
5	.182	.584	.384	.381	.387	.378	.382
Ave.	.185	.586	.385	.386	.385	.386	.386

Table 23. Selection x linkage means of the variability among selected parents expressed as fractions of the variance in the unselected population with environmental variance

Gen.	0.0	0.1	1.0	1.1	...
	$1 + K$				
1	.146	.142	.175	.183	.161
2	.139	.140	.176	.174	.157
3	.125	.142	.164	.179	.152
4	.140	.131	.176	.168	.154
5	.127	.133	.173	.167	.150
Ave.	.135	.138	.173	.174	.155
	$1 + \overline{K}$				
1	.192	.186	.597	.610	.396
2	.186	.187	.586	.599	.389
3	.181	.186	.578	.584	.382
4	.182	.183	.573	.571	.377
5	.178	.185	.596	.571	.382
Ave.	.183	.187	.588	.585	.386

Table 24. Analyses of variance of the variability among selected parents as fractions of the variance in the unselected population with no environmental variance (arcsin transformation)

SOV	d.f.	$\_1 + K$	$1 + \overline{K}$
Generation	4	.01353[c]	.00516
Selection	1	.03032[c]	26.91874
Population	1	.00017	.00109
Linkage	1	.03605	.00710
SXP	1	.00005	.00035
SXL	1	.02976[c]	.01185[a]
PXL	1	.00046	.00113
SXPXL	1	.00002	.00404
GXS	4	.01697[c]	.00533
GXP	4	.00175	.00051
GXL	4	.00539	.00328
GXSXP	4	.00911[b]	.00443
GXSXL	4	.01002[b]	.00210
GXPXL	4	.00149	.00353
GXSXPXL	4	.00560	.00879[a]
ERROR	600	.00239	.00272

[a] $P < .05$

[b] $P < .01$

[c] $P < .001$

Table 25. Analyses of variance of the variability among selected parents as fractions of the variance in the unselected population with environmental variance (arcsin transformation)

SOV	d.f.	$1 + K$	$1 + \overline{K}$
Generation	4	.00266	.01011[a]
Selection	1	.22288[b]	31.32980[b]
Population	1	.00176	.00002
Linkage	1	.00051	.00025
SXP	1	.00045	.00245
SXL	1	.00005	.00001
PXL	1	.00017	.00106
SXPXL	1	.00087	.00000
GXS	4	.00010	.00478
GXP	4	.00216	.00279
GXL	4	.00265	.00244
GXSXP	4	.00116	.00113
GXSXL	4	.00061	.00456
GXPXL	4	.00059	.00058
GXSXPXL	4	.00400	.00027
ERROR	600	.00165	.00243

[a] $P < .01$

[b] $P < .001$

Table 26. Analysis of variance of achieved heritability--no environmental variance

SOV	d.f.	ΔG ÷ Reach
Generation	4	.00014
Selection	1	.00004
Population	1	.00034
Linkage	1	.00010
SXP	1	.00011
SXL	1	.00012
PXL	1	.00004
SXPXL	1	.00000
GXS	4	.00020
GXP	4	.00005
GXL	4	.00042
GXSXP	4	.00039
GXSXL	4	.00039
GXPXL	4	.00022
GXSXPXL	4	.00004
ERROR	600	.00032

Table 27. Means of the ratios of components of variance to the values expected

Gen.	000	001	010	011	100	101	110	111
	S/E(S)							
1	0.919	1.007	1.003	1.073	0.775	1.022	1.015	0.904
2	1.032	1.006	0.811	0.996	1.278	0.957	1.121	1.190
3	0.913	1.035	0.934	0.892	1.066	1.062	0.808	0.857
4	1.060	0.952	1.113	0.942	1.010	0.994	0.929	0.931
5	1.036	1.022	1.053	1.124	0.884	1.106	0.807	0.938
Ave.	0.992	1.004	0.982	1.005	1.002	1.028	0.936	0.964
	D/E(D)							
1	1.107	1.040	0.927	0.946	1.051	1.000	1.002	0.982
2	1.028	1.117	1.097	1.051	0.960	1.022	0.968	0.979
3	1.056	0.940	1.012	0.986	1.006	1.001	1.026	1.017
4	0.928	0.997	1.004	1.034	0.998	0.987	1.021	1.032
5	1.060	1.044	1.007	1.004	1.000	0.964	1.017	0.988
Ave.	1.036	1.027	1.009	1.004	1.003	0.995	1.007	0.999
	W/E(W)							
1	1.029	0.998	0.947	0.993	1.002	0.985	1.003	0.986
2	1.289	1.543	1.252	1.536	1.463	1.224	1.495	1.266
3	1.343	1.545	1.132	1.526	1.244	1.296	1.200	1.329
4	1.145	1.507	1.362	1.501	1.281	1.319	1.206	1.297
5	1.271	1.430	1.371	1.447	1.230	1.321	1.117	1.305
Ave.	1.216	1.405	1.213	1.401	1.244	1.229	1.204	1.236

Table 28. Main effect means of the ratios of components of variance to the values expected

Gen.	0..	1..	.0.	.1.	..0	..1	...
	S/E(S)						
1	1.000	0.929	0.931	0.999	0.928	1.002	0.965
2	0.961	1.137	1.068	1.030	1.061	1.037	1.049
3	0.944	0.948	1.019	0.872	0.930	0.962	0.946
4	1.017	0.966	1.004	0.979	1.028	0.955	0.991
5	1.059	0.934	1.012	0.981	0.945	1.047	0.996
Ave.	0.996	0.983	1.007	0.972	0.978	1.000	0.989
	D/E(D)						
1	1.005	1.009	1.049	0.964	1.022	0.992	1.007
2	1.073	0.982	1.032	1.024	1.013	1.042	1.028
3	0.998	1.012	1.001	1.010	1.025	0.986	1.005
4	0.991	1.009	0.978	1.023	0.988	1.012	1.000
5	1.029	0.992	1.017	1.004	1.021	1.000	1.010
Ave.	1.019	1.001	1.015	1.005	1.014	1.006	1.010
	W/E(W)						
1	0.992	0.994	1.004	0.982	0.995	0.990	0.993
2	1.405	1.362	1.380	1.387	1.375	1.393	1.384
3	1.386	1.268	1.357	1.297	1.230	1.424	1.327
4	1.379	1.276	1.313	1.342	1.249	1.406	1.327
5	1.380	1.243	1.313	1.310	1.247	1.376	1.312
Ave.	1.308	1.229	1.273	1.264	1.219	1.318	1.269

Table 29. Selection x linkage means of the ratios of components of variance to the values expected

Gen.	0.0	0.1	1.0	1.1	...
	S/E(S)				
1	0.961	1.040	0.895	0.963	0.965
2	0.922	1.001	1.200	1.074	1.049
3	0.923	0.964	0.937	0.959	0.946
4	1.086	0.947	0.969	0.963	0.991
5	1.045	1.073	0.845	1.022	0.996
Ave.	0.987	1.005	0.969	0.996	0.989
	D/E(D)				
1	1.017	0.993	1.026	0.991	1.007
2	1.062	1.084	0.964	1.001	1.028
3	1.034	0.963	1.016	1.009	1.005
4	0.966	1.016	1.010	1.009	1.000
5	1.034	1.024	1.008	0.976	1.010
Ave.	1.023	1.016	1.005	0.997	1.010
	W/E(W)				
1	0.988	0.995	1.002	0.986	0.993
2	1.270	1.540	1.479	1.245	1.384
3	1.238	1.535	1.222	1.313	1.327
4	1.254	1.504	1.244	1.308	1.327
5	1.321	1.439	1.174	1.313	1.312
Ave.	1.214	1.403	1.224	1.233	1.269

Table 30. Analyses of variance of the ratios of actual components of variance to their expected values.

SOV	d. f.	S/E(S)	D/E(D)	W/E(W)
Generation	3	.22831	.01830	.12842[c]
Selection	1	.00012	.07223	1.29122[c]
Population	1	.96779	.00895	.00594
Linkage	1	.01123	.00032	1.98204[c]
S X P	1	.17245	.00346	.02354
S X L	1	.00686	.00011	1.52950[c]
P X L	1	.09547	.00469	.01170
S X P X L	1	.04486	.00094	.05152
G X S	3	.49355	.08434	.05281
G X P	3	.10661	.02234	.04622
G X L	3	.18111	.03626	.18461[c]
G X S X P	3	.19550	.01380	.13764[c]
G X S X L	3	.21676	.01954	.34339[c]
G X P X L	3	.21817	.02725	.06489[a]
G X S X P X L	3	.06293	.01290	.06390[a]
Error	600	.23103	.03352	.02210

[a] $P < .05$

[b] $P < .01$

[c] $P < .001$

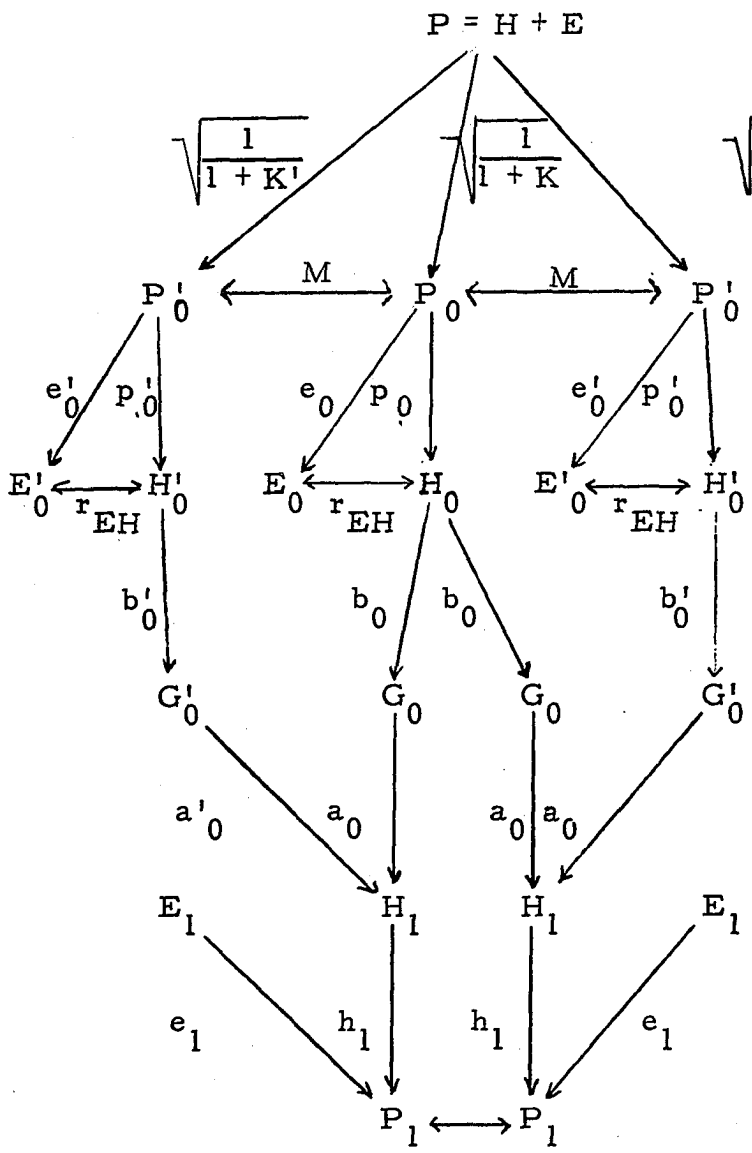
Table 31. Means of potential genic variances and actual genic variances and the ratios of these means--no environmental variance

Gen.		000	001	010	011	100	101	110	111
0	$2\Sigma_i p_i q_i$	20.00	20.00	19.96	19.96	20.00	20.00	19.96	19.96
	$\sigma^2 H_n$	19.78	19.66	19.85	19.92	19.87	20.10	19.91	20.09
	Ratio	1.011	1.017	1.006	1.002	1.007	0.995	1.003	0.994
1		19.33	19.32	18.98	18.93	19.78	19.76	19.42	19.44
		12.13	11.63	11.05	11.77	15.50	15.88	15.74	15.15
		1.594	1.661	1.718	1.608	1.276	1.244	1.234	1.283
2		17.79	17.92	17.00	17.18	19.30	19.27	18.61	18.60
		9.22	10.30	8.01	10.20	15.77	14.36	16.15	13.89
		1.930	1.740	2.122	1.684	1.224	1.342	1.152	1.339
3		15.60	15.90	13.86	15.01	18.47	18.56	17.32	17.61
		7.24	8.96	5.14	8.77	14.29	13.62	14.06	13.17
		2.155	1.775	2.696	1.712	1.293	1.363	1.232	1.337
4		12.54	13.48	10.90	12.57	17.46	17.63	15.59	16.49
		4.56	7.64	4.00	7.41	13.17	12.86	12.23	12.25
		2.750	1.764	2.725	1.696	1.326	1.371	1.275	1.346
5		9.99	10.78	8.73	10.02	16.24	16.51	13.65	15.34
		3.38	6.26	3.13	6.08	11.67	12.17	10.16	11.31
		2.956	1.722	2.789	1.648	1.392	1.357	1.344	1.356
6		9.89	10.75	8.48	9.84	16.15	16.48	13.19	15.05
		4.19	8.64	3.49	8.03	14.52	13.82	11.91	13.48
		2.360	1.244	2.430	1.225	1.112	1.192	1.107	1.116
7		9.92	10.70	8.25	9.63	16.08	16.42	12.93	14.74
		4.78	9.67	3.88	8.58	15.05	16.07	10.74	13.49
		2.075	1.107	2.126	1.122	1.068	1.022	1.204	1.093

Table 32. Means of potential genic variances and actual genic variances and the ratios of these means--environmental variance

Gen.		000	001	010	011	100	101	110	111
0	$2\Sigma_i p_i q_i$	20.00	20.00	19.96	19.96	20.00	20.00	19.96	19.96
	$\sigma^2 H_n$	19.72	20.21	19.95	19.68	20.16	20.06	20.05	20.28
	Ratio	1.014	0.990	1.000	1.014	0.992	0.997	0.996	0.984
1		19.61	19.61	19.27	19.27	19.87	19.86	19.49	19.49
		15.75	15.82	15.27	15.57	17.48	17.71	17.77	17.56
		1.245	1.240	1.262	1.238	1.137	1.121	1.097	1.110
2		18.78	18.75	18.05	18.14	19.59	19.58	18.87	18.86
		13.65	14.31	12.41	13.65	18.36	17.10	17.33	16.20
		1.376	1.310	1.454	1.329	1.067	1.145	1.089	1.164
3		17.55	17.58	16.35	16.79	19.15	19.17	18.03	18.20
		11.83	13.69	9.82	12.77	17.39	16.74	16.62	15.61
		1.484	1.284	1.665	1.315	1.101	1.145	1.085	1.166
4		16.11	16.10	14.06	15.29	18.54	18.62	16.97	17.37
		10.13	12.42	7.76	12.09	16.29	16.32	14.53	14.71
		1.590	1.296	1.812	1.265	1.138	1.141	1.168	1.181
5		14.47	14.44	11.93	13.62	17.86	17.96	15.71	16.60
		8.06	11.15	6.37	10.69	15.43	15.39	13.12	14.02
		1.795	1.295	1.873	1.274	1.157	1.167	1.197	1.184

APPENDIX C: FIGURES



$$p_0^2 = \frac{h^2(1+K)}{1+Kh^2}$$

$$b_0^2 = \frac{1}{2}\left[\frac{1+Kh^2}{1+Kh^2/2}\right]$$

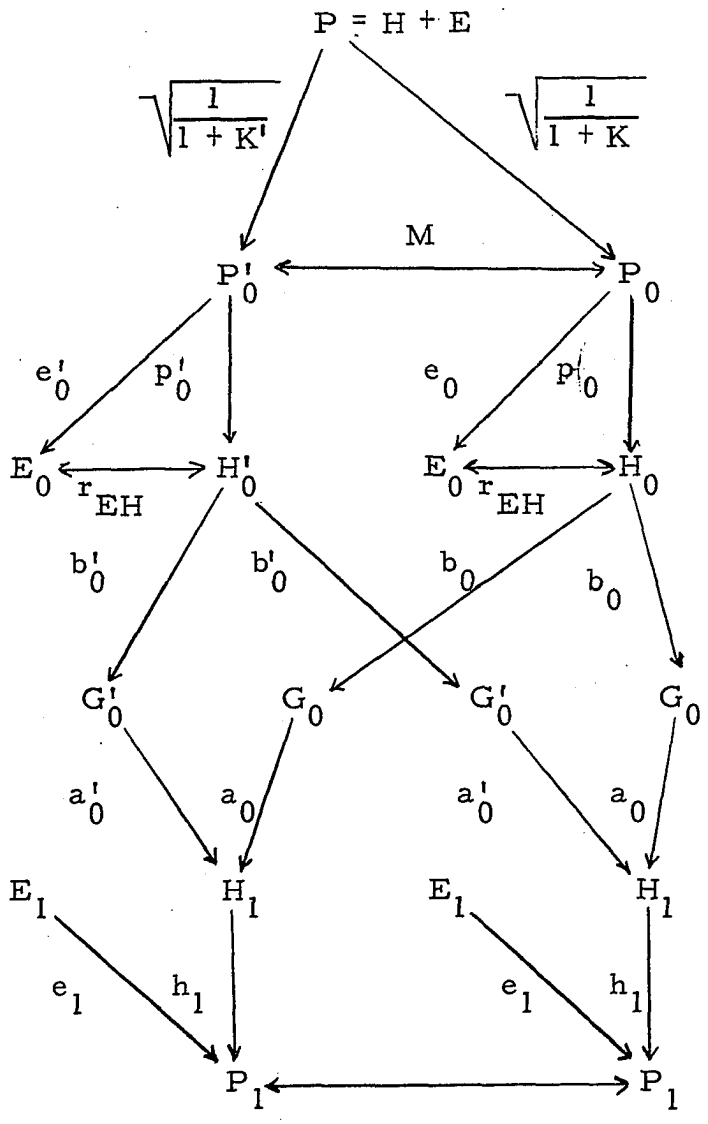
$$a_0^2 = \frac{1}{2}\left[\frac{1+Kh^2/2}{1+\bar{K}h^2/2}\right]$$

$$h_1^2 = h^2\left[\frac{1+\bar{K}h^2/2}{1+\bar{K}h^4/2}\right]$$

$$r_{PHS} = b_0^2 a_0^2 h_1^2 + 2M\, p_0'^2 b_0'^2 a_0'^2 h_1^2$$

$$= \frac{h^2}{4}\left[\frac{1+Kh^2}{1+\bar{K}h^4/2}\right] \qquad \text{when } M = 0$$

Figure 1. The correlation between paternal half sibs when parents have been selected such that the variance among selected female parents as a fraction $(1 + K')$ of the variance in the unselected parent population and the variance among selected male parents is a fraction $(1 + K)$.



$$p_0^2 = \frac{h^2(1+K)}{1+Kh^2}$$

$$b_0^2 = \frac{1}{2}\left[\frac{1+Kh^2}{1+Kh^2/2}\right]$$

$$a_0^2 = \frac{1}{2}\left[\frac{1+Kh^2/2}{1+\bar{K}h^2/2}\right]$$

$$h_1^2 = h^2\left[\frac{1+\bar{K}h^2/2}{1+\bar{K}h^4/2}\right]$$

$$r_{FS} = a_0^2 b_0^2 h_1^2 + a_0'^2 b_0'^2 h_1^2 + 2a_0 a_0' b_0 b_0' h_1^2 p_0 p_0' M$$

$$= \frac{h^2}{2}\left[\frac{1+\bar{K}h^2}{1+\bar{K}h^4/2}\right] \quad \text{when } M = 0$$

Figure 2. The correlation between full sibs when parents have been selected such that the variance among selected female parents is a fraction $(1 + K')$ of the variance in the unselected parent population and the variance among selected male parents is a fraction $(1 + K)$.

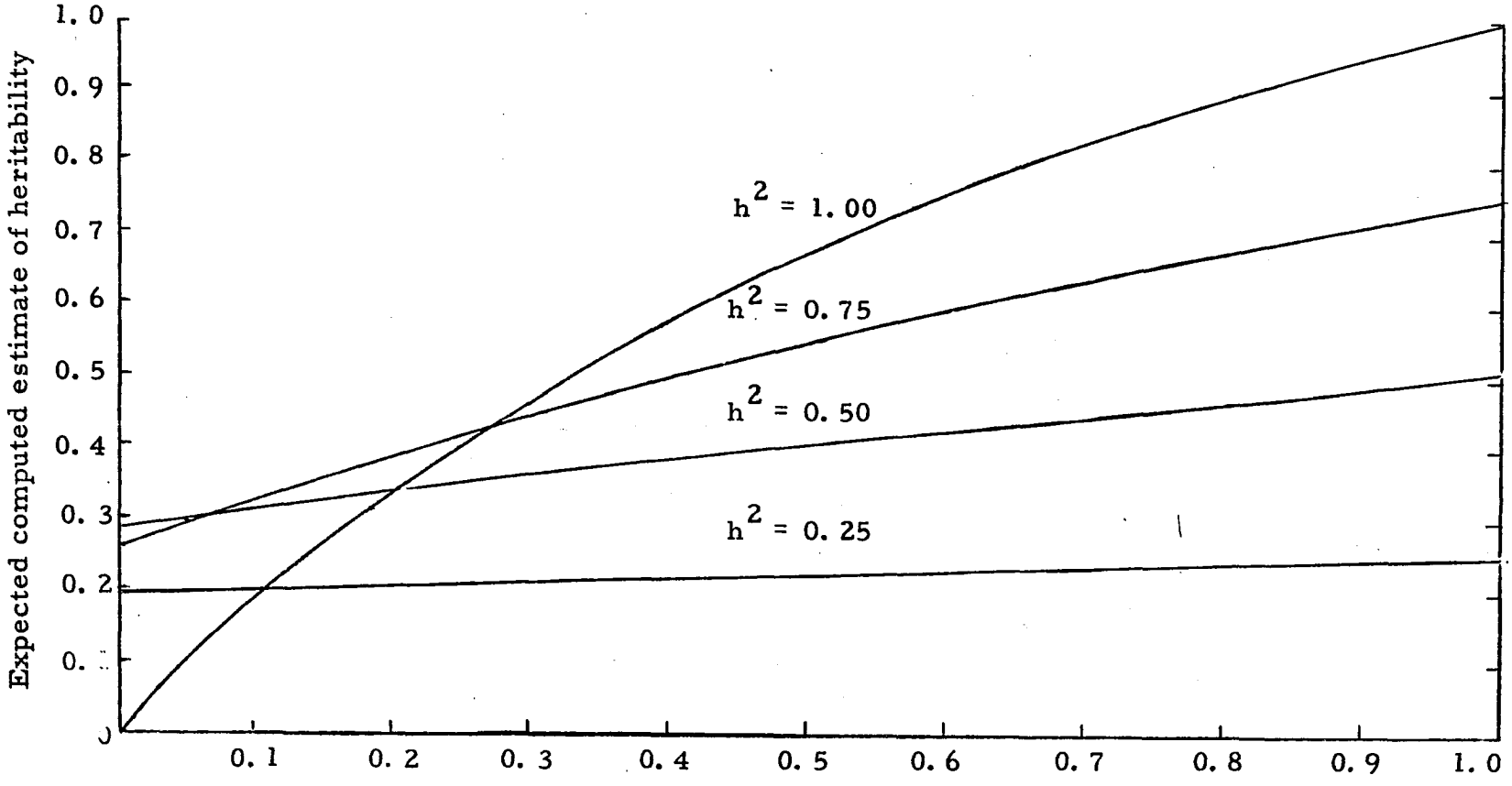


Figure 3. Expected computed estimates of heritability, when the variation among parents is reduced by selection (intensity of selection the same in both sexes).

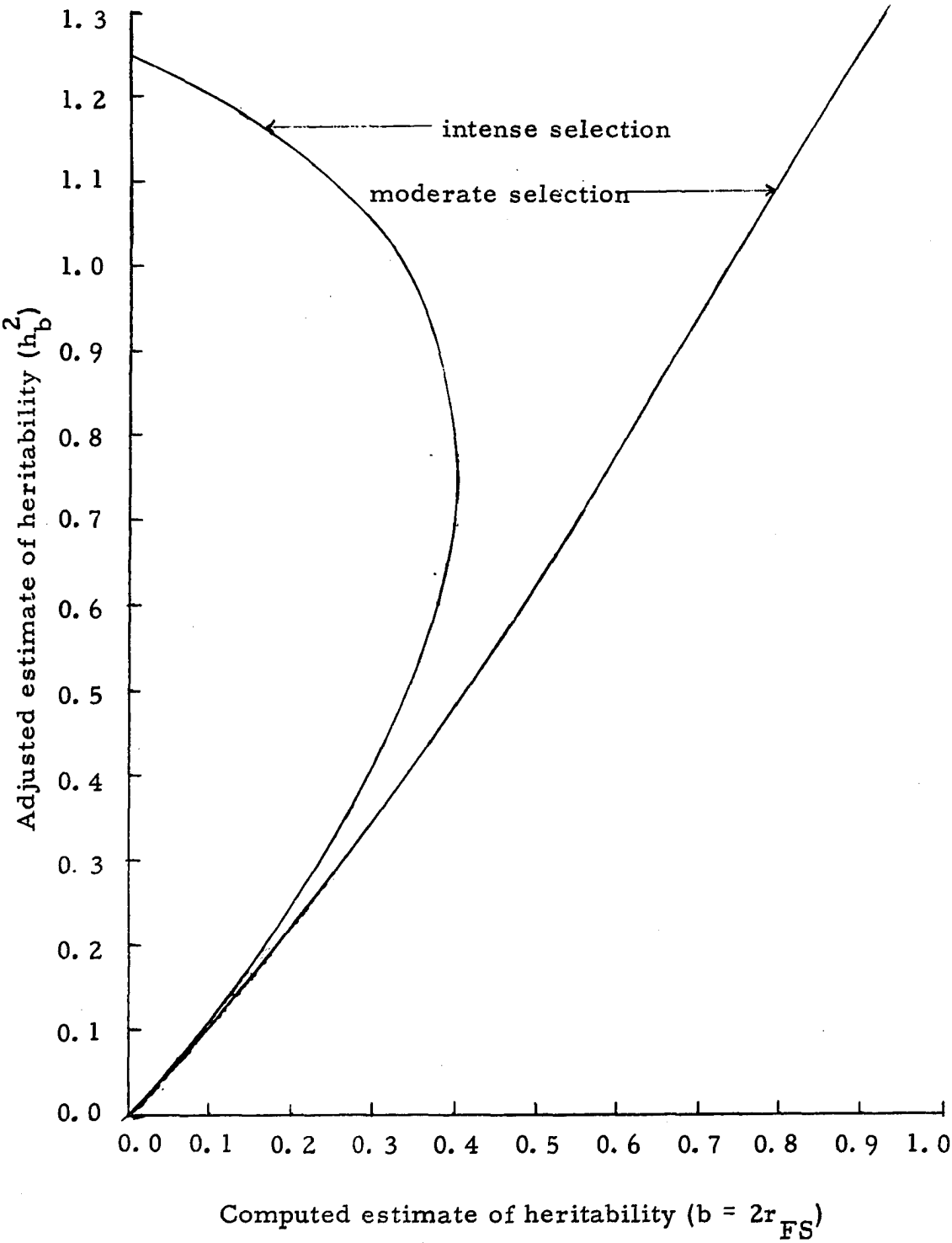


Figure 4. Graphs of the adjusted estimates of heritability (h_b^2) for a range of computed full sib estimates ($b = 2r_{FS}$). The fraction of variance remaining among selected parents is taken to be $1 + \bar{K} = 0.194$ for intense selection and $1 + \bar{K} = 0.592$ for moderate selection.

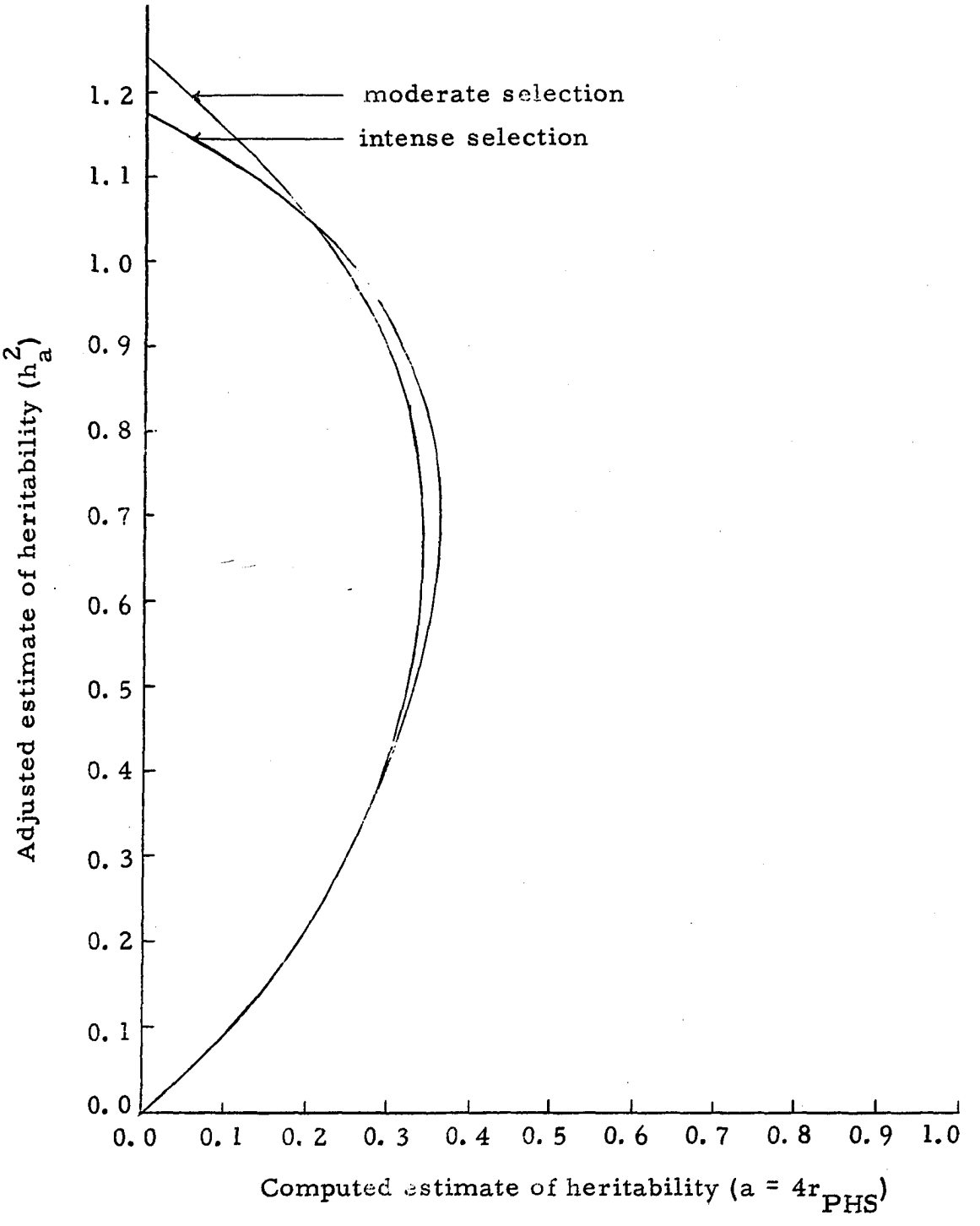


Figure 5. Graphs of the adjusted estimates of heritability (h_a^2) for a range of computed paternal half sib estimates ($a = 4r_{PHS}$). The fraction of variance remaining among selected parents is taken to be $1 + K = 0.156$ and $1 + \bar{K} = 0.194$ for intense selection and $1 + K = 0.184$ and $1 + \bar{K} = 0.592$ for milder selection.

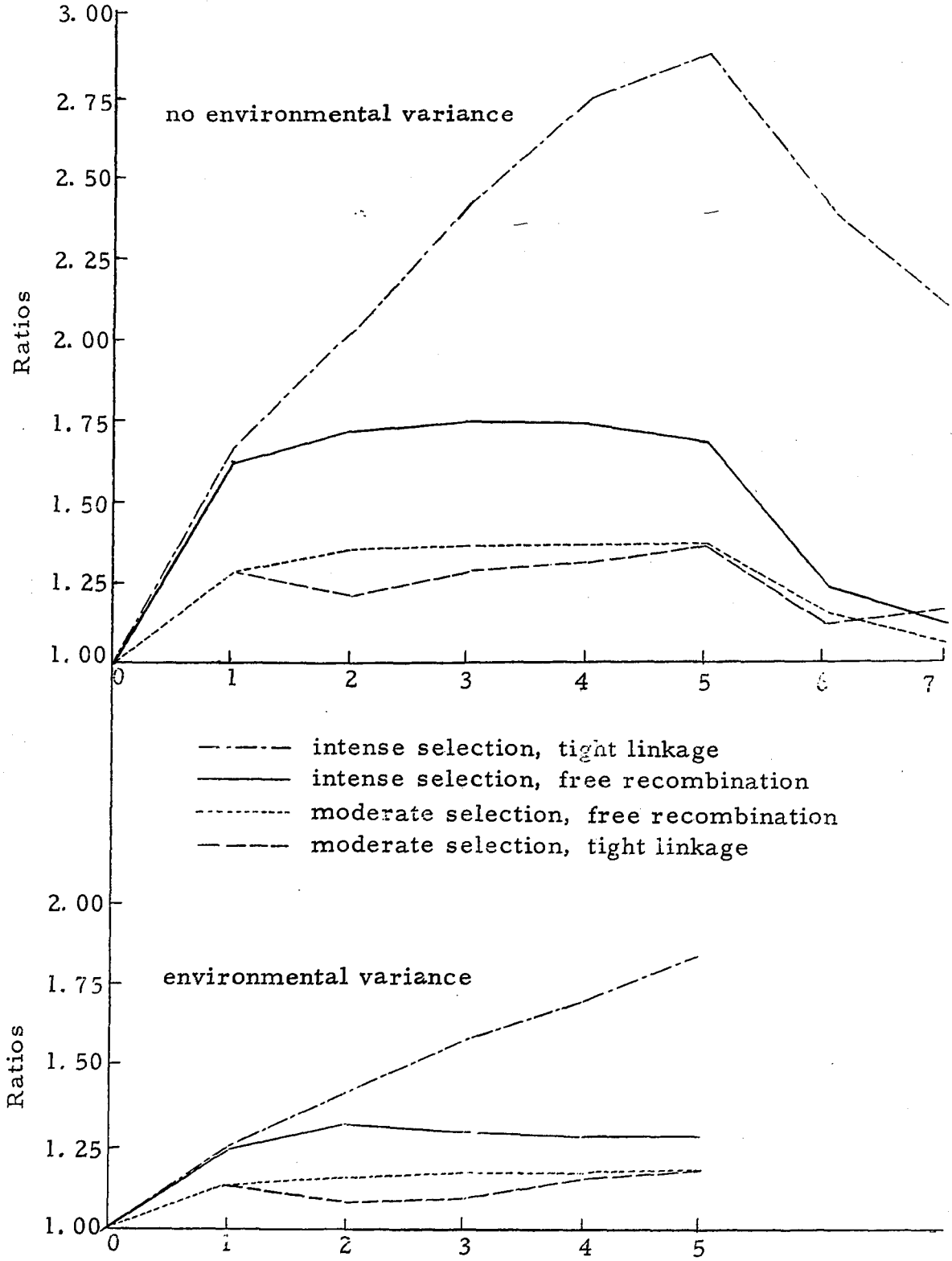


Figure 6. Ratios of potential genic variance to actual genic variance by levels of selection and linkage (over population sizes)